FLUID BALANCE
IN SURGICAL PRACTICE

These are much deeper waters than I had thought

Sherlock Holmes in "The Reigate Squires"

Fluid Balance in Surgical Practice

L. P. LE QUESNE
D.M. (Oxon.), F.R.C.S. (Eng.)

Assistant Director, Department of Surgical Studies, Middlesex Hospital

SECOND EDITION



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PREFACE TO THE SECOND EDITION

WITH the passage of years our understanding of the fluid and electrolyte problems seen in surgical practice increases steadily, and much that was new a few years ago has now been incorporated into the fabric of routine surgical care. In this new edition an attempt has been made on the one hand to bring the monograph up to date with advances made since its first publication, and on the other to correct a certain disproportion between its various sections, resulting in part from the conditions of the original prize essay. With these aims in view, the entire text has been revised, and the sections dealing with the metabolic response to surgery and its practical implications have been largely re-written. In conformity with their increasing importance, Nitrogen and Calorie problems, whilst strictly outside the scope of this work, receive more detailed consideration.

A completely new section is included on the problems of fluid balance in infants and children undergoing surgery. In this highly specialised subject, in modern practice increasingly dealt with by pædiatricians, exact attention to the details of treatment is of even greater importance than in adults. I am greatly indebted to my friend, Dr. B. W. Webb, for his most valuable contribution, which is based on an extensive, practical experience in this field.

I am indebted to Mr. Rodney Maingot and Messrs. H. K. Lewis & Co., Ltd., for their permission to reproduce, from the book *The Management of Abdominal Operations*, Figs. 1–4, 24, 31, 33 and 34 and Tables I and III; to Masson et Cie (Paris) for permission to reproduce Fig. 7; to the Editor of the *Annals of the Royal College of Surgeons* for permission to reproduce Figs. 5, 6, 8, 11–14, 16, 17, 27 and 28; to the Editor of the *Lancet* for permission to reproduce Fig. 9; and to the Editor of the *Proceedings of the Royal Society of Medicine* for permission to reproduce Figs. 10 and 21.

L. P. Le Q.

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PREFACE TO THE FIRST EDITION

THIS monograph is based on the Moynihan Prize Essay for 1953, entitled *The Management of Fluid and Electrolyte Exchange in Surgical Patients*. The conditions of this essay specify that particular attention will be paid to original work, and this consideration has in great part determined the content of the monograph, in particular the initial section dealing with the metabolic changes following operation. It was decided not to alter this method of presentation, as it was felt that discussion of a concrete series of observations provided a firm introduction for a consideration of the whole problem of electrolyte disturbances following operation.

In the pages that follow an attempt is made to discuss, without undue elaboration, the theoretical background to fluid and electrolyte exchanges, and in addition to describe practical methods for dealing with these problems as they arise in clinical surgery. Accordingly, wherever possible, points of importance are illustrated by observations made personally, and in addition the monograph contains an appendix of annotated case-histories, illustrating the management of various derangements of fluid balance.

I am most grateful to the Council of the Association of Surgeons of Great Britain and Ireland for permitting me to publish my essay in this modified form. The larger part of the work on which this essay is based was carried out whilst I was working on the Surgical Unit, Middlesex Hospital, and I am grateful to Mr. P. B. Ascroft for the opportunities and facilities I was allowed. I am also indebted to Mr. E. W. Riches and Mr. W. R. Winterton for allowing me to study some of their cases and to publish details in this monograph. I have throughout worked in close collaboration with my colleague Dr. A. A. G. Lewis and I am most grateful to him for his advice and encouragement. I am also most grateful to Mr. G. L. S. Pawan, B.Sc., without whose skilled technical assistance this work would not have been possible. Finally, I must express my thanks to Dr. Walter Somerville for his advice and assistance with the electrocardiographic records

L. P. Le Q.

February 1954.

PHYSIOLOGICAL CONSIDERATIONS

INTRODUCTION

MODERN surgical practice is notable not only for the growing range and magnitude of the operations performed, but also for the diminishing mortality and morbidity after established procedures. Our ability to achieve both these objects is due in great measure to an increasing control over all aspects of the patient's response to surgery, and recent years have seen a markedly increased concentration on the accessory forms of treatment and management, which together combine to enable operations hitherto fatal to be carried out with success. In particular, renewed interest has been shown in fluid and electrolyte problems, and our increasing understanding of the physiology and pathology of the body fluids is a major contribution to surgical endeavour.

The magnitude of many modern operations places such a strain upon the patient's economy that the smallest deviation from normality may prove disastrous, and it was this increasing necessity to control the patient's metabolism which stimulated renewed surgical interest in fluid and electrolyte problems. In addition, two other factors have greatly affected surgical thought and practice in relation to this subject. First, the introduction within recent years of the milli-equivalent as a common unit of measurement has contributed enormously to the general understanding of the subject. Secondly, and of greater importance, has been the development of the flame-photometer, by use of which sodium and potassium analyses of the body fluids can now be made accurately and rapidly. It would be easy to over-emphasise the contribution of these two technical advances, but it is doubtful whether much recent work of great value could have been performed in their absence. In addition, the use of a flame-photometer makes routine sodium and potassium estimations so simple that accurate control of electrolyte disturbances is now possible in all surgical departments.

Fluid and electrolyte disturbances consist essentially in alterations in normal physiological processes, so that an understanding of the abnormal states must start from a consideration of the normal distribution and exchange of water and salt before proceeding to a discussion of changes produced by surgical operations and pathological processes. Many of the concepts and ideas involved in these considerations are complex. Although modern research has resulted in much simplification in our ideas, it has also brought out many

aspects of the subject must be considered together, and an appendix of case histories is included to illustrate the complete problem as it must be faced and managed in clinical practice.

UNITS OF MEASUREMENT: MILLI-EQUIVALENTS AND MILLI-OSMOLS

Until recent years custom has decreed that the concentration of the electrolytes in the body fluids shall be expressed in mgm. per cent.—that is to say, in terms of the absolute weight of the substances in solution. However, this disregards the fact that chemical substances react together not by absolute weight but by equivalent weight. Thus, in the formation of sodium chloride one equivalent weight of Na (23 gm.) reacts with one equivalent weight of Cl (35·5 gm.) to produce one equivalent weight of NaCl (58·5 gm.). If, instead of a system based on absolute weights, a unit of measurement is used which neglects these differences in absolute weight and expresses the concentration of electrolytes in terms of equivalent weight, the information provided becomes highly significant, and gives an accurate picture of the chemical structure of the fluid concerned. The unit of measurement found convenient for this purpose is the milli-equivalent (mEq) and milli-equivalent per litre (mEq/l.), one milli-equivalent being defined as one-thousandth of the equivalent weight of a substance expressed in grammes. The advantage of the use of mEq/l. as opposed to mgm. per cent. is clearly shown in Fig. 1, and there can be no doubt that the introduction of this unit of measurement greatly simplifies the understanding of human fluid and electrolyte physiology and pathology: custom alone prevents its more rapid introduction into routine clinical usage.

The conversion of milligrammes per cent. into milli-equivalents per litre can be accomplished by the following formula:

$$\frac{\text{mgm. per cent.} \times 10 \times \text{valency}}{\text{atomic weight}} = \text{milli-equivalents per litre.}$$

The factors, derived from this equation, for the conversion of the common plasma constituents are shown in Table I. Table II, listing the normal plasma concentration of these constituents expressed in both units of measurement, shows how the use of mgm. per cent. masks the actual structure of this fluid.

Neither of these units of measurement already mentioned gives ready information concerning one other important property of body fluids, namely their osmotic force. The osmotic force exerted by a substance in solution depends directly on the number of particles in solution, irrespective of their valency, and to express the osmotic structure of a solution the unit of measurement used is milli-osmols

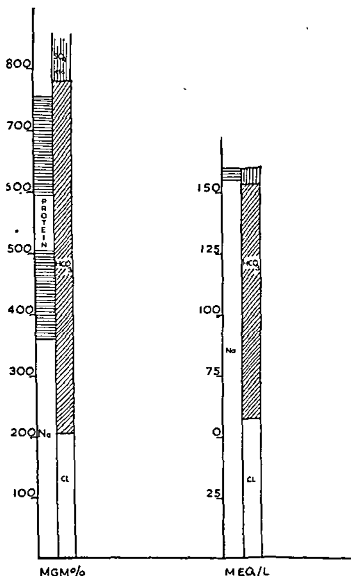


FIG. 1.—Stick graphs showing an analysis of pancreatic juice expressed in mgm. per cent and mEq/l The fluid contains nearly three times as much sodium as chloride, but this fact is obscured by the use of mgm. per cent, whereas the use of mEq/l demonstrates clearly the essential structure of the fluid.

problems of which we were previously unaware, and which still await solution. As a result it is impossible to make a comprehensive review of the subject without posing as many problems as existed previously, save by neglecting inconvenient facts and observations. Further, modern work has opened up so many new aspects of the subject that a detailed consideration of them all demands a breaking down of the whole into artificial divisions. However, in clinical practice all

of water in the body bears a constant relationship, not to the total body mass, but to the lean tissue mass, with the result that fat people contain relatively less water than thin. Edelman *et al.* (1952) investigated this point in detail. They found that the total body water, measured by the deuterium oxide dilution technique, was highest in infancy (77 per cent.) and falls to a mean of 59 per cent. between the ages 1–9 years. After puberty a distinct sex difference becomes apparent, males containing relatively more water than females, probably because of the greater amount of fat in females. They write: "The importance of total body fat in relation to total body water is apparent when one considers that the lean body mass is constant in composition, and that the relation of the lean body mass to the total body water is probably also constant in the normal. Of the three major constituents of body composition (fat, lean tissue and water), the only one which varies independently of the other two in a healthy, normal person is fat. Therefore, the body habitus is a prime determinant in the relative content of water and fat." Nevertheless, bearing these limitations and variations in mind, the figure 70 per cent. remains a sound, clinical guide to the total water content of the body.

The body water does not exist as a single continuous fluid medium, but is divided into two main portions—namely the intracellular and the extracellular water—the anatomical extent of these divisions or spaces being descriptively obvious. Of these two spaces the intracellular is by far the larger, containing, in an average adult of 70 Kg., 35 litres of water, whilst the extracellular space contains only 15 litres. The extracellular space is further divided into the intravascular space (i.e. blood plasma) and the extravascular or interstitial space (Fig. 2). Of the 15 litres of water in the extracellular space 12 litres lie in the interstitial space and 3 litres form the plasma water. These divisions of body water do not, of course, form distinct, rigidly divided units, and there is a constant, copious exchange between them; however, they do form distinct functional units, and the concept of these divisions of the body water is essential to an understanding of electrolyte physiology.

These three divisions of the body water—namely the intracellular space, the interstitial space and the intravascular space—differ not only in their size, but also in their electrolyte components (Figs. 2 and 3), and furthermore they are separated from one another by membranes with specific properties. The intracellular water contains protein; its main cation, or base, is potassium, and its main anion, or acid radical, is phosphate. Interstitial water contains no protein, its main cation is sodium, and its main anion chloride, with bicarbonate a subsidiary but most significant anion. These two spaces are separated from one another by the cell membrane, which is freely

TABLE I

$\frac{\text{Na mgm. per cent.}}{23}$ = Na mEq/l.

$\frac{\text{K mgm per cent.}}{3{\cdot}9}$ = K mEq/l.

$\frac{\text{Ca mgm. per cent.}}{2}$ = Ca mEq/l.

$\frac{\text{Cl mgm per cent}}{35}$ = Cl mEq/l.

$\frac{CO_2 \text{ Combining Power Vols. per cent.}}{2{\cdot}2}$ = Bicarbonate mEq/l.

Factors for the conversion of mgm. per cent. into mEq/l.

TABLE II
NORMAL PLASMA VALUES

Substance	*mgm per cent.*	*mEq/l.*
Na . .	310–335	135–145
K . . .	15–22	3 9–5 2
Ca .	9–11	4·5–5·5
Cl .	342–378	97–107
CO_2 Combining Power	53–72 Vols. per cent.	24–32

In discussing plasma chloride levels it is a common, but essentially confusing, practice to estimate the actual plasma chloride and then express it as sodium chloride, the normal range being 600–650 mgm. per cent.

per litre (mgm. per cent. × 10 ÷ atomic weight). In the case of monovalent ions such as Na, Cl and K, their milli-equivalent and milli-osmolar value will be identical, but in the case of divalent ions, such as Ca and Mg, the chemical milli-equivalence is twice the osmolar value. The unit of measurement milli-osmols is rarely, if ever, used in hospital practice, but demands recognition owing to its not infrequent use in the literature on electroltye problems.

NORMAL DISTRIBUTION AND COMPOSITION OF THE BODY FLUIDS

In health, approximately 70 per cent. of the body substance by weight consists of water. However, this percentage must not be considered fixed, but rather as varying somewhat according to age, sex, build, etc. As postulated by Behnke (1942), it appears that the amount

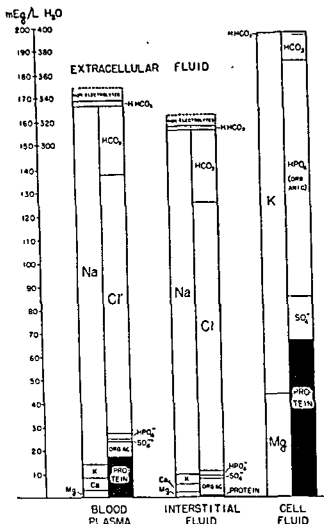


FIG. 3.—Stick graphs showing the composition of cell fluid, interstitial fluid and blood plasma (from Gamble, 1950). The non-electrolytes consist of glucose, nitrogenous waste products, etc., etc., which are carried around within the body by the fluids of which the electrolytes form the permanent structure.

of fluid and electrolyte across the vascular membrane, water and salts being ejected from the vascular space by the hydrostatic pressure in the arterial capillaries and drained back into the venous capillaries by the osmotic force of the plasma proteins.

Over and above their absolute volume the most important property of the body fluids is their tonicity, and the maintenance of these fluids at approximately normal concentration is essential to life. Accordingly the amount of the fluid in the body, and to a great extent its distribution between the intra- and extracellular spaces, is essentially dependent on the quantity of electrolytes available, and

permeable to water but relatively impermeable to electrolytes, with the result that the osmotic forces set up by the substances in solution on either side of this membrane largely control the volume of the two spaces. In the intravascular space the electrolyte constituents (Table II) are the same as in the interstitial space, the only difference between the two being the presence of the plasma proteins in the former. The vascular membrane separating these two spaces is permeable to both water and electrolytes, but not to protein. Though anatomically separate, these two divisions of the extracellular space form a functional unit, as there is a constant large-scale exchange

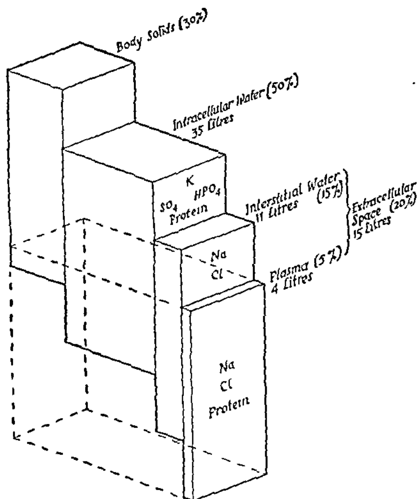


FIG. 2.—Diagram illustrating the distribution of water within the body, together with the size and main constituents of the three fluid compartments. The dotted line represents the total body volume, with its constituent portions pushed up like toy bricks from a box. The figures in brackets show the percentage of the total volume represented by each portion.

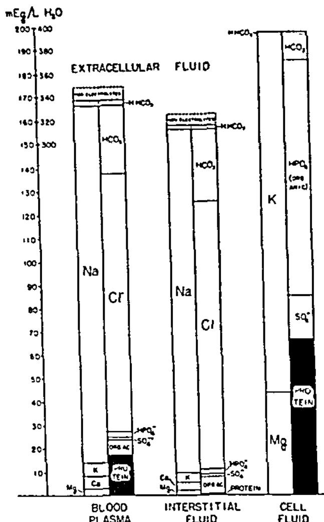


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any alteration in fluid volume should primarily be considered not as an alteration in water, but in electrolytes. It is for this reason that sodium is of such vital importance to the body economy. Whereas potassium is within limits replaceable by sodium, and chloride is within wide limits replaceable by bicarbonate, there is no other cation capable of replacing sodium in the extracellular space. Accordingly, a loss of sodium from the body will lead to a loss of water, and a gain of sodium to a gain of water, both changes resulting from the overriding physiological consideration of the maintenance of approximately normal tonicity and concentration of salts in the body fluids.

By virtue of their osmotic activity the proteins in the intracellular and intravascular fluids play an important part in the distribution of the body water, though, as emphasised by Abbott (1946), their osmotic force in relation to that of the electrolytes is insufficient to influence the total amount of water within the body. This action of the proteins in controlling the distribution of water is particularly important in respect of the part played by the plasma proteins in maintaining the correct distribution of water between the intravascular and interstitial spaces. Though the osmotic force exerted by the plasma proteins is only 2 milli-osmols per litre (Gamble, 1950), it has been calculated that but for their presence a man would lose all his plasma water into the interstitial space within 10 seconds. Furthermore, the plasma proteins play a part not only in the distribution of the water within the body, but also of the electrolytes. Reference to Fig. 3 will show that the total milli-equivalence of both the intracellular and intravascular fluids slightly exceeds that of the interstitial fluid. The slight excess of intravascular electrolytes is due to the fact that the vascular membrane is impermeable to the plasma proteins, which, though only weakly ionised, yet carry an electrical charge, so that some interstitial electrolytes must pass into the intravascular space in accordance with Donnan's Law, to restore the electrical equilibrium on either side of the membrane.

The reason for the higher concentration of electrolytes in the intracellular fluid is not so clear, but is probably accounted for by the fundamental processes responsible for the maintenance of the intracellular protoplasm. So far, the distribution of water and electrolytes within the body has been described on the basis of the classical theory of chemical and osmotic equilibrium between the body compartments. However, it is now becoming clear that other mechanisms are involved. The work of Robinson (1950), Terner, Eggleston and Krebs (1950) and others has shown that the maintenance of normal, intracellular tonicity is a dynamic process, which demands an expenditure of body energy, and which breaks down in the absence of oxygen and certain substrates. It appears that these

mechanisms are capable of shifting water and electrolytes against an osmotic gradient, and are adjusted to maintain the cells slightly hypertonic in respect of interstitial fluid. Despite these reservations the classical theory has been retained, both in this section and that dealing with water and salt depletion, as it provides a convenient and essentially rational basis for clinical work.

In consideration of the distribution of body water, two further simple facts need emphasis. First, whilst the intravascular space is the only one whose contents can readily be analysed, such an analysis will give information concerning the constituents of the whole extracellular space, as the plasma water differs from the interstitial only in its protein content. Thus a fall in the plasma sodium or urea concentration signifies a similar fall throughout the whole 15 litres of the extracellular space, not just in the 3 litres of intravascular water. Secondly, the large size of the interstitial compared to the intravascular space means that the former constitutes an extensive and vitally important reservoir which may be drawn upon in an emergency to maintain the plasma volume.

NORMAL INTAKE AND OUTPUT

In normal life an individual's intake of water and electrolytes varies considerably from day to day, and the output of each is regulated by the body to maintain the normal volume and composition of the body fluids. This balance is not strictly accurate over short periods of time, but is remarkably accurate over a period of several days (see Gamble, 1951), and must be regarded not as a static phenomenon, but as a dynamic process aimed at preserving constant the *milieu intérieure* of the body. Despite the fact that in normal health the intake varies considerably, none the less by an examination of the obligatory and desirable daily losses from the body by all normal routes it is possible to calculate the optimal daily intake of water and electrolytes.

Water is normally lost from the body by four routes, namely from the respiratory passages, from the skin, in the fæces, and in the urine.

Respiratory Losses. Atmospheric air is relatively dry, whereas that in the alveoli of the lungs is saturated with water vapour. This saturation of the inspired air takes place mainly in the upper air passages, and normally some 400 ml. of water are lost from the body each 24 hours in this process, though in abnormally dry climates or in conditions causing a raised respiratory rate this figure may be substantially larger.

Cutaneous Losses. Water is lost from the skin in two distinct ways—there is a constant diffusion of water vapour from the skin,

and in addition there is a loss of both water and some salt in the sweat. The amount of fluid lost by both these routes is enormously variable, depending on the environmental temperature and humidity, and on the body temperature and activity. A man in normal health loses some 600–1000 ml. of water from the skin each 24 hours, together with a little salt in the sweat, the electrolyte content of which is about one-third that of plasma. In disease or as a result of strenuous activity the losses from the skin may be much larger, and Moyer (1952) states that the maximum rate of loss from the lungs and skin combined is 5·12 per cent. of the body weight per hour—that is to say, 3·5 litres per hour in a 70 Kg. man.

In view of the fact that, save under unusual circumstances, the salt loss in the sweat is insufficient to be considered in clinical calculations, it is convenient for these purposes to group together the entire losses from the skin and lungs. Unfortunately there is no satisfactory term to describe this loss. There are cogent objections to the use of the three most obvious terms—namely "insensible loss," "inevitable loss" and "extrarenal loss". Whilst fully aware that the term is not descriptively accurate, throughout this monograph the term "insensible loss" will be used to include loss of water vapour from the lungs and skin, together with sweat loss. In normal health this insensible loss amounts to 1000–1500 ml. per day. In thus considering together the losses from the skin and lungs, two vital facts need stressing, namely that, apart from the sweat, this loss consists of pure water, and further it is inevitable—that is to say, it is a continuous process, not under the control of the mechanisms which regulate water balance, and accordingly should be considered as having first demand on such water as is available to the body.

Fæces. The normal adult loses some 60–150 ml. of water daily from the bowel, and with it small quantities of electrolytes. In certain diseases losses from this route may be much larger.

Urine. Water loss by the above routes is essentially inevitable, and not under the control of the body's water-regulating mechanisms. Such, however, is not true of the water loss by the kidneys, as it is by variation in the daily volume of urine that the water balance of the body is maintained. After the requirements of the insensible loss have been met, any water remaining superfluous in the body is available to the kidneys for excretion as urine water. In order to prevent nitrogenous retention, the kidneys also have the task of removing from the body at least 35 gm. of waste products per 24 hours, as well as any excess electrolytes. The amount of water required by the kidneys for this purpose depends essentially on their concentrating power. If both kidneys are healthy and can concentrate to a specific gravity of 1·032, 500 ml. of water will suffice for this purpose, but, as shown by Fig. 4, if there is any impairment of

the kidneys' concentrating power, the minimal daily urine volume necessary to prevent nitrogenous retention rises rapidly. It is generally agreed that 1500 ml. per 24 hours is the optimal urine output, as this not only allows plenty of scope for any depression in

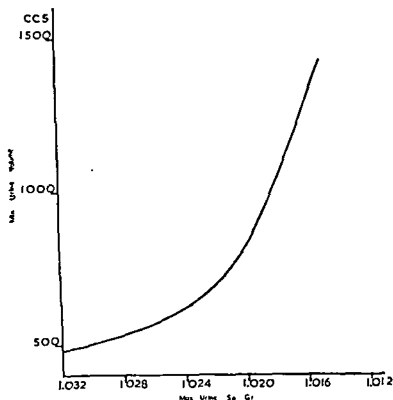


FIG 4.—Graph showing the relationship between the maximum urinary specific gravity and the minimal daily urine volume necessary to prevent nitrogenous retention (modified from Marriott, 1947, calculated from the figures of Newburgh and Lashmet, 1932 and 1933).

kidney function and for the excretion of increased amounts of nitrogen and electrolytes, but also relieves the kidney from the necessity of excreting highly concentrated urine.

The water intake of the body is derived from two different sources, namely endogenous and exogenous.

Endogenous water is that released by the metabolic activities of the body, and amounts to up to 500 ml. per 24 hours. Normally this water consists of the "water of oxidation" formed during the oxidation of ingested food, but during starvation there is the additional "preformed water" released by the breakdown of body tissue (see Coller and Maddock, 1933; Newburgh, 1951).

Exogenous water is all that either drunk or ingested in solid food; this supply varies greatly from day to day, but usually amounts to

and in addition there is a loss of both water and some salt in the sweat. The amount of fluid lost by both these routes is enormously variable, depending on the environmental temperature and humidity, and on the body temperature and activity. A man in normal health loses some 600–1000 ml. of water from the skin each 24 hours, together with a little salt in the sweat, the electrolyte content of which is about one-third that of plasma. In disease or as a result of strenuous activity the losses from the skin may be much larger, and Moyer (1952) states that the maximum rate of loss from the lungs and skin combined is 5·12 per cent. of the body weight per hour—that is to say, 3·5 litres per hour in a 70 Kg. man.

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tutes a reasonable basic daily allowance, and that 2 gm. is the minimum requirement to prevent depletion. Information with respect to optimal potassium requirements is even more scanty; but it would appear that the normal intake is somewhere between 3–4 gm. K per 24 hours (Lans *et al.*, 1952)—that is to say, the amount of potassium contained in approximately 3·5–10·0 gm. of KCl (50–150 mEq). Probably 100 mEq (7·5 gm. KCl) represents a satisfactory basic intake, and there is some evidence (*vide infra*) that an intake substantially less than this causes a compensatory sodium retention. The mixed diet of a healthy person contains both salt and potassium in amounts greater than those specified above, and the excess is excreted in the urine, but the amounts specified provide an ample basic intake provided there are no abnormal losses.

Apart from this overt exogenous daily exchange, there is a much larger internal turnover of water and electrolytes each day, disturb-

TABLE IV

THE NORMAL VOLUME OF DIGESTIVE JUICES SECRETED PER 24 HOURS BY AN ADULT

Source	*Volume*
Saliva	1500 c.c.
Gastric secretion	2500 c.c.
Bile	500 c.c.
Pancreatic juice	700 c.c.
Secretion of intestinal mucosa	3000 c.c.
TOTAL	8200 c.c.
Normal Plasma Volume	3500 c.c.

(From Gamble, 1950)

ances of which can easily lead to gross imbalance in the external exchange. The constant and large-scale exchange of extracellular fluid between the vascular and interstitial compartments has already been mentioned, 73 per cent. of the blood water exchanging with the extracellular fluid every minute (Reaser and Burch, 1945). The formation of œdema in heart failure or renal disorders is a derangement of this mechanism severe enough to affect the external exchanges of the body. Of a similar nature is the daily formation of some 170 litres of glomerular filtrate, of which all but 1–2 litres is reabsorbed into the body in the renal tubules; in acute tubular

2–3 litres per 24 hours, of which from about one-third to one-half is contained in the food eaten.

For clinical purposes this daily balance sheet can be considerably simplified, without significant loss of accuracy, by ignoring the fæcal losses and endogenous water supply. This leaves the optimal basic water intake as 3000 ml. per 24 hours, to provide 1500 ml. for insensible loss and 1500 ml. for urine water (Table III). Many

TABLE III

NORMAL DAILY FLUID BALANCE

Intake		*Output*	
By mouth	3000 ml.	Insensible loss Urine	1500 ml. 1500 ml.
TOTAL	3000 ml.	TOTAL	3000 ml.

For the sake of simplicity in clinical usage, endogenous water and water loss in the fæces can be ignored without introducing significant inaccuracies.

authorities quote a lower figure than this as the optimal water intake, but this is probably unwise. Save under especial circumstances, 3000 ml. per day can not be a dangerously large intake, and yet provides plenty of water for any increase in the insensible loss, or any increase in the urine volume necessitated either by some diminution in kidney function or an increased solute load. Further, this figure has additional advantages in connection with the replacement of abnormal losses. Over a large series of cases, and in very varying circumstances, this amount has been administered (with one important exception, pp. 47 and 48) as the basic daily water requirement, with excellent results. However, it is to be borne in mind that these figures refer to a normal-sized adult, and with very small adults it is often wise to restrict the intake to 2·0 or 2·5 litres per 24 hours. (See special section for requirements in children.)

Unfortunately the daily requirements of salt and of potassium cannot be calculated in a fashion similar to those of water. There is a small obligatory daily loss of salt in the sweat and fæces, and of potassium in the urine and fæces, but the normal daily intake of both salt and potassium far exceeds these losses, the excess being excreted in the urine. This urinary loss is not, of course, essential, but there are obvious advantages in providing an intake sufficient to maintain the urinary concentration of these substances within normal limits. In regard to salt it is generally considered that 5 gm. per day consti-

tutes a reasonable basic daily allowance, and that 2 gm. is the minimum requirement to prevent depletion. Information with respect to optimal potassium requirements is even more scanty; but it would appear that the normal intake is somewhere between 3–4 gm. K per 24 hours (Lans *et al.*, 1952)—that is to say, the amount of potassium contained in approximately 3·5–10·0 gm. of KCl (50–150 mEq). Probably 100 mEq (7·5 gm. KCl) represents a satisfactory basic intake, and there is some evidence (*vide infra*) that an intake substantially less than this causes a compensatory sodium retention. The mixed diet of a healthy person contains both salt and potassium in amounts greater than those specified above, and the excess is excreted in the urine, but the amounts specified provide an ample basic intake provided there are no abnormal losses.

Apart from this overt exogenous daily exchange, there is a much larger internal turnover of water and electrolytes each day, disturb-

TABLE IV

THE NORMAL VOLUME OF DIGESTIVE JUICES SECRETED PER 24 HOURS BY AN ADULT

Source	*Volume*
Saliva	1500 c.c.
Gastric secretion	2500 c.c.
Bile	500 c.c.
Pancreatic juice	700 c.c.
Secretion of intestinal mucosa .	3000 c.c.
TOTAL	8200 c.c.
Normal Plasma Volume . .	3500 c.c.

(From Gamble, 1950)

ances of which can easily lead to gross imbalance in the external exchange. The constant and large-scale exchange of extracellular fluid between the vascular and interstitial compartments has already been mentioned, 73 per cent. of the blood water exchanging with the extracellular fluid every minute (Reaser and Burch, 1945). The formation of œdema in heart failure or renal disorders is a derangement of this mechanism severe enough to affect the external exchanges of the body. Of a similar nature is the daily formation of some 170 litres of glomerular filtrate, of which all but 1–2 litres is reabsorbed into the body in the renal tubules; in acute tubular

necrosis (*vide infra*) this internal exchange is so grossly disturbed that death may occur, at different stages of the disease, from either overhydration or depletion, unless specific care is taken to prevent these disasters. But of far greater importance in surgical practice is the daily turnover of fluid and electrolytes in the intestines. Table IV shows the average daily volume of the various secretions discharged into the alimentary tract; of this 8 litres (i.e. an amount greater than the circulating blood volume), normally all but 100–200 ml. are reabsorbed into the body. Furthermore, these intestinal juices *consist, not of water, but of a fluid containing electrolytes in a* concentration varying in the different secretions, but essentially equal to that of extracellular fluid. A moment's consideration of these facts reveals how easily a disturbance of this normal intestinal exchange can lead to rapid and severe depletion of both water and electrolytes, and, indeed, in conditions such as intestinal obstruction, this depletion often presents the most urgent therapeutic aspect of the case.

The Mechanism and Control of Normal Balance

The physiological mechanisms by which the human body maintains control of water and electrolyte balance are complex and imperfectly understood. In a sense, the water balance is controlled by the antidiuretic hormone of the posterior pituitary, secretion of which causes an increased reabsorption of water by the renal tubes and so a diminution in the urine volume. However, as shown by Verney (1946), the secretory activity of the posterior pituitary is regulated by special cells (the osmoreceptors, probably located in the supraoptic nucleus) which are sensitive to alterations in the osmotic pressure of the plasma crystalloids, especially sodium and chloride. Accordingly, the posterior pituitary mechanism should *primarily be regarded as the guardian of the osmotic pressure of the* body, a function which it performs by varying the water excretion (Lewis, 1952). In other words, the posterior pituitary does not control the *absolute* amount of water in the body, but the amount of water relative to the amount of crystalloids, especially sodium.

It is now becoming apparent that, separate from this control of osmolarity, there is a control of water excretion responsive to volume changes, the receptor organs of this mechanism probably being situated in the heart, great vessels and mediastinum. Henry and Gauer (1951) showed that, in dogs, the urine flow was halved following an hæmorrhage of 20 per cent. of the estimated blood volume, and doubled following a plasma transfusion of an equivalent amount. Similarly, Welt and Orloff (1951) have shown that the infusion of iso-osmotic solutions of albumin in normal human subjects causes a striking diuresis. In a later paper, Gauer *et al.* (1954) showed that,

gs, periods of constant negative pressure breathing caused a
stently increased urine flow, and suggested that this diuresis
due to a redistribution of blood, with engorgement of the
cic viscera, and was an example of one of the many conditions
ich, in normal subjects, an engorgement of the intrathoracic
latory organs, no matter how induced, coincides with an in-
ed urinary flow. They suggested that stretch receptors in the
onary circulation and/or chambers of the heart and great vessels
represent the receptor part of a mechanism linking hæmostatic
ts with urine excretion in the control of plasma volume. The
re of this linkage is unknown, but it appears that the afferent
way of the reflex lies in the vagus nerve (Henry and Pearce,
).

ie antidiuretic hormone has, in man, no effect on the excretion
ectrolytes (Chalmers, Lewis and Pawan, 1951). Basically, both
um and potassium balance are probably controlled by adreno-
ical hormones. It was thought that, in their turn, the secretion
hese hormones was regulated by the anterior pituitary gland.
s the administration of cortisone causes a diminished sodium
increased potassium excretion by the kidneys, and ACTH pro-
es similar effects by stimulation of the adrenal cortex. However,
owing the isolation of aldosterone (electrocortin) by Tait and
pson (see Simpson & Tait, 1955, for a full summary of their
k) it has become apparent that other mechanisms are involved.
influence of aldosterone on the renal excretion of electrolytes
ar more potent than that of any other known adrenocortical
act, and it seems probable that it is in fact the hormone normally
trolling such excretion. But it appears that the secretion of aldos-
ne is not influenced by the pituitary. Axelrad *et al.* (1954)
wed that the administration of ACTH caused no rise in the
nary output of sodium-retaining corticoids, and similarly,
nmerman *et al.* (1956) have shown that the human adrenal does
require the participation of ACTH in the control of sodium con-
vation. The work of Borst and de Vries (1950) suggests that the
retion of adrenocortical hormones may be dependent upon the
diac output and venous pressure, and further there is evidence
t the secretion of aldosterone is dependent upon the dietary
ake of sodium (Garrod *et al.*, 1956). However, it is not yet clear
w all the various mechanisms described are integrated together,
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for the operative loss, and from 126-280 gm. for the 4 hours following operation. The authors concluded, "The total fluid lost (i.e. including blood loss) during the period of operation and recovery by a patient on whom a laparotomy is performed will average close to a litre. Much greater losses occurred in certain cases in which there was marked sweating or blood loss." And further, "In the first 12 cases the urine output is less than 15 per cent. of the total water lost (i.e. including blood loss) during the entire time. In the last 6 patients the urine output rose from 22–50 per cent. of the total fluid loss." Apart from the immediate operative period these workers also showed (Coller and Maddock, 1940) that the insensible loss of a group of "surgical sick patients" during hot, humid weather varied between 2068 ml. and 5034 ml. per 24 hours.

These striking figures of Coller and Maddock were obtained in a climate hotter and more humid than that usually encountered in this country, at a time when theatre coverings were heavier than those usually in use now, and when closed-circuit anæsthesia was not generally in use. Undoubtedly they serve to draw attention to a potential hazard, but our own observations suggest that nowadays the losses on the day of operation are not so great. The data obtained in a series of cases observed for other purposes (Le Quesne and Lewis, 1953) also allowed calculations to be made of the insensible loss. The average figure for this insensible loss per 24 hours in a group of 10 patients over the 3 days prior to a partial gastrectomy was 1050 ml., ranging from 760–1240 ml. These patients, all males, were afebrile, spent part of the day in bed and part sitting about the ward, and observations were made at all times of the year. In 5 of these cases it was also possible to calculate the insensible loss during the 24 hours starting at the time the pre-operative sedative was injected, and surprisingly the average figure was only 1070 ml., the range being 700–1500 ml. Admittedly the method of calculation of this latter loss was not entirely satisfactory. Further, the exposure of viscera during gastrectomy is not great, and in none of these patients did any obvious sweating occur, so that in many cases the insensible loss is likely to be greater than this. Nevertheless, these figures suggest that the insensible loss over the period of operation need not be excessive provided that the patient is not overheated, and that in this country under normal circumstances the figure of 1500 ml. should be sufficient to cover insensible loss even on the day of operation.

METABOLIC CHANGES AFTER OPERATION

Of greater importance than this increase in the insensible loss is the complex metabolic disturbance which occurs after operation,

EFFECTS OF OPERATION ON WATER AND ELECTROLYTE BALANCE

THE performance of any surgical operation necessarily leads to certain changes in the normal mechanisms of water and electrolyte balance. In general, the magnitude of these changes varies directly with the severity of the operation. Further, it is generally true to say that in the majority of cases these changes are not sufficiently dramatic to modify the course of recovery, provided intakes are kept within reasonable limits. However, it is also true that in all cases these changes narrow the limits within which the intake of water and electrolytes can safely be varied; that in certain cases they cause clinically obvious alterations in normal physiological processes; and in addition that the superimposition of these changes upon the complex problems of depletion and dehydration may give rise to situations which are extremely difficult to control and correct. For all these reasons, quite apart from more general academic considerations, an understanding of these changes is essential to the correct and confident management of fluid administration in the post-operative period. The majority of these changes are part of a complex metabolic response to trauma (i.e. surgical operation) which is not yet completely understood, but there is, in addition, a simple physical factor which will be considered first.

INSENSIBLE LOSS DURING OPERATION

Operations of any magnitude are usually accompanied by an increase in the insensible loss, due to the raised temperature in the operating theatre, to the exposure of viscera at operation, and at times to the considerable degree of sweating which may follow operation. This point was investigated by Coller and Maddock (1932), who carried out studies on 18 patients, based essentially on careful weighing before and after operation; in 12 of these cases the patients wore the heavy theatre clothing and coverings then in use at that hospital, and for comparison the remaining 6 cases had much lighter coverings. Their results showed that in the group of 12 cases heavily covered the total water loss from the skin and lungs during operation ranged from 97–705 gm. (i.e. equivalent to ml.), and in the immediate 4 hours after operation 217–828 gm., making a total loss over both periods of well over 1 litre in many cases. In the 6 more lightly covered patients the figures ranged from 92–347 gm.

they received after operation these patients would, under normal circumstances, be expected to lose weight at the rate of 0·2-0·5 Kg (½-1 lb.) per 24 hours, so that any weight gain under these circumstances must indicate an increase in body water. The metabolic data obtained in these studies has been charted in the usual way (Fig. 5).

Water Excretion

Following operation there is a marked impairment of the kidneys' ability to excrete water, lasting for some 24-36 hours after an operation of the severity of a sub-total gastrectomy. Figure 6 shows the urine output of 8 patients for three consecutive 24-hour periods,

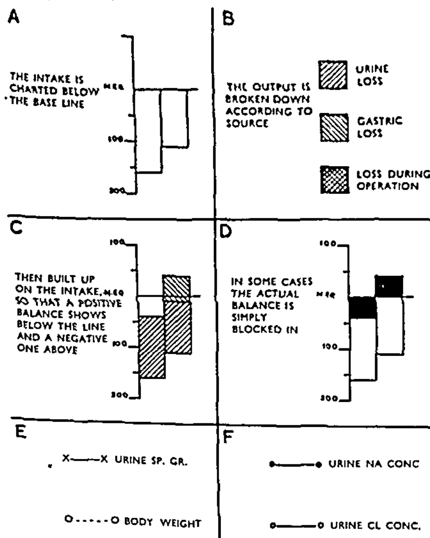


Fig. 5.—Chart illustrating the method used to record balance studies. Note that a positive balance (retention) shows below the base-line, and a negative balance above it

involving not only the bodily exchanges of water and salt, but also potassium and nitrogen equilibrium. Essentially this post-operative disturbance, which, in general, varies in magnitude and duration directly with the severity of the operation, results in a temporary impairment of the body's normal ability to excrete water and salt, together with an increased mobilisation and excretion of potassium and nitrogen. This metabolic disturbance is a normal physiological response of the organism to trauma, and as such should be regarded as beneficial. In the great majority of patients undergoing surgical operations the effects of this disturbance are so slight and transient that they demand no attention. However, in those undergoing major procedures the metabolic disturbance causes marked changes which, if neglected and mis-managed, can cause serious complications. In the first few days after operation it is the alterations in water and electrolyte excretion which are most likely to lead to acute difficulties, but later the changes in nitrogen (protein) metabolism may cause as serious, if less acute, troubles. (See section on nitrogen and calorie problems.)

As long ago as 1905 Pringle, Maunsell and Pringle observed the diminished urine flow occurring after operation, but it was the work of Coller, Dick and Maddock (1936) on post-operative salt and water retention that stimulated modern research into the problem. Since then a great deal of work has been done on the subject, and in general the effects of the disturbance are accurately known, though much concerning its causation and significance remains obscure. It is now clear that the effects of the post-operative metabolic response are very complex, and it is only possible to understand the response by dissecting apart its various components and considering them separately. Much of the description that follows is based on an investigation carried out at the Middlesex Hospital by my colleague, Dr. A. A G. Lewis, and myself, and reported in detail elsewhere (Le Quesne and Lewis, 1953; Le Quesne, 1954). In this investigation balance studies were carried out on 24 patients, all save 2 of whom underwent sub-total gastrectomy for benign peptic ulcer. The essential basis of these observations was the use of a technique by which a constant intake of water and electrolytes was maintained for several days before and after operation, the only variation being that after operation the intake was given intravenously, contained no protein, and provided, in the form of glucose, only 600 cals. per 24 hours. The intake of water and electrolytes that these patients received (usually 4 0 litres of water and 160–170 mEq Na per 24 hours) was designed to reveal clearly any changes taking place after operation, and not as the intake desirable at this time. Balance studies were carried out in the usual manner and the patients were weighed daily. It is important to stress that on the calorie intake

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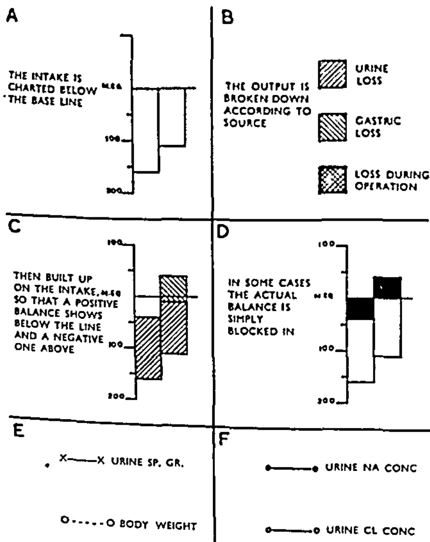


FIG. 5.—Chart illustrating the method used to record balance studies. Note that a positive balance (retention) shows below the base-line, and a negative balance above it.

the operation in each case taking place at the start of the second period. Each of these patients received each 24 hours 4 litres of water and 160–170 mEq Na, with, in addition, 500 ml. citrated blood during operation, except for the man undergoing herniorrhaphy, who throughout received a smaller, oral intake. It can be clearly seen that during the 24 hours immediately following operation the urine output is markedly diminished, with a return to, or above, pre-operative levels on the ensuing day. That this falling-off in urine output is not due to increased insensible loss during the operation is shown by the body-weight of these patients. Figure 7 shows that each one gained weight considerably during the 24 hours after operation, despite the fact that all save the last were, during this period and for 2–4 days thereafter, on intravenous fluids providing only 600 calories (150 gms. glucose) each 24 hours. The average gain in weight of these 8 cases during the day of operation was 1·7 Kg. (3·8 lb.), and even when allowances have been made for insensible loss and the expected weight loss from tissue breakdown, this must represent a retention of over 1 litre of water in each case. The 2 cases undergoing colectomy and herniorrhaphy respectively are of additional interest, in that they show that this phenomenon of impaired water excretion is not due to the operation of gastrectomy *per se*, and the latter case, studied by a different technique giving oral fluids throughout, also shows that it is not due to the change-over from oral to intravenous feeding.

This temporary impairment of water excretion is a primary disturbance of water balance, independent of the salt intake and balance over this period. The eight cases illustrated in Figs. 6 and 7 all received salt throughout the periods recorded, and all showed a markedly positive sodium balance during the 24 hours after operation (*vide infra*). Studies were made on other patients who received no salt on the day of operation, save for that contained in 0·5 litre of citrated blood (Na 60 mEq: Cl 35 mEq), and data from 2 of these patients are shown in Fig. 8 It will be seen that both were in negative sodium balance on this day (even without taking into account the estimated operative loss) yet both, receiving 4 litres of water per 24 hours, show a definite weight gain in the 24 hours after operation, indicating clearly that during this period there had been an impaired excretion and retention of water, despite a negative sodium balance.

The period of impaired water excretion with diminished urine flow is accompanied by characteristic changes in the urine, which has a high specific gravity and raised electrolyte concentration (Figs. 6 and 7). In all the cases so far illustrated, and in fact in the majority of patients, there was on the day after operation (O + 1) a raised urine flow, with a fall in specific gravity and electrolyte con-

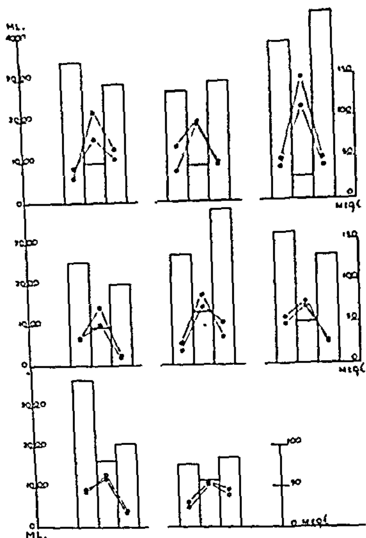


FIG. 6.—Graphs showing the urine volume (blocks), urine sodium concentration (dots) and urine chloride concentration (circles) for 3 consecutive 24-hour periods in 8 patients, each of whom underwent operation at the start of the second period. The upper 6 of the cases illustrated underwent a partial gastrectomy, the lower two sigmoid colectomy and herniorrhaphy respectively (see Fig. 7). The diminished urine flow following operation is clearly shown: this is accompanied by a raised urine electrolyte concentration. (See text for intake of these cases.)

centration. This was sometimes accompanied by a loss of part or all of the weight gain due to retained water, though in some cases this was masked by further salt and water retention (*vide infra*). However, in 2 cases the specific gravity of the pooled urine on the day after operation remained greater than 1·010, indicating a more prolonged period of impaired water excretion. Such observations on pooled 24-hour urine specimens give only gross information

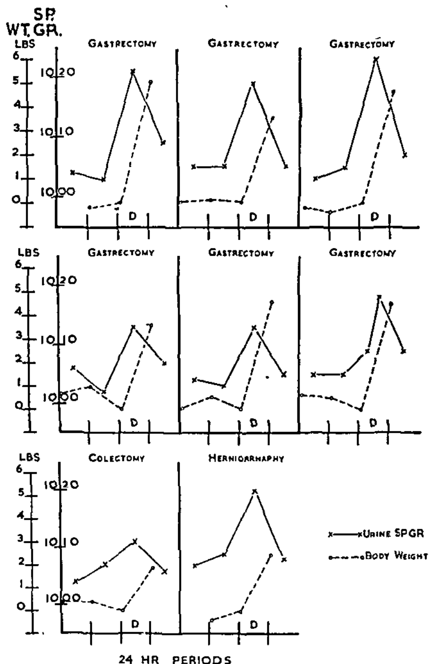


FIG. 7.—Body weight and urine specific gravity data from the 8 cases illustrated in Fig 6 All cases showed a distinct weight gain during the 24 hours immediately following operation (O), and this was accompanied by a rise in urine specific gravity which fell to pre-operative levels in the ensuing period

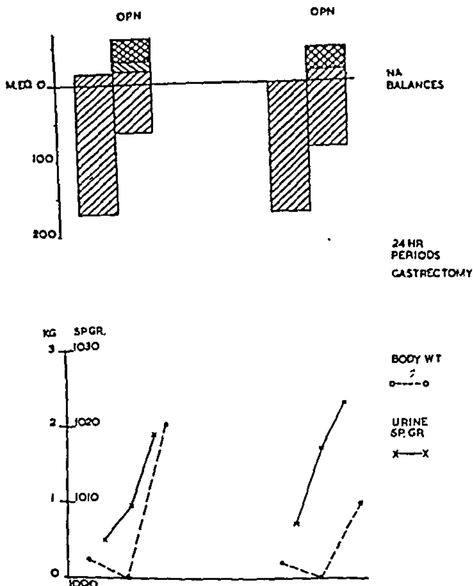


FIG. 8.—Data from 2 cases receiving no salt on the day of operation, except for that in 500/ml. of citrated blood. Both cases were in negative sodium balance on the day of operation (OPN), but both show a weight gain over this period as a result of retaining water.

concerning a phenomenon which appears to have a clear-cut start and finish. Accordingly, investigations were performed on a further series of cases in which, owing to the presence of an indwelling urethral catheter, it was possible to study 4-hourly urine specimens (Le Quesne, 1954). These studies showed that at the start of an operation there is an abrupt reduction of urine flow and a rise in specific gravity, and that these changes usually last some 24–36 hours after

operation, when they cease as abruptly as they began (Fig. 9). However, it is to be noted that in exceptional cases the diminution in urine flow may last up to 72 hours after operation, and, though very rare, this occurrence is of great importance on account of the resultant danger of water intoxication (*vide infra*).

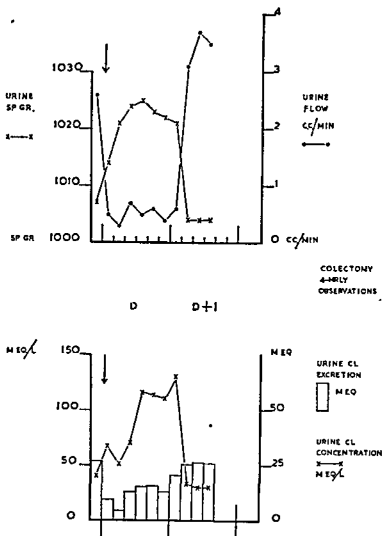


FIG. 9—Graphs showing the urine volume (ml./min.), urine specific gravity, urine Cl concentration and absolute chloride output in a patient undergoing sigmoid colectomy, obtained by observations on 4-hourly urine specimens from an indwelling catheter. Fluid and electrolyte intake constant throughout period of observation and for 48 hours previously (4 litres of water, 170 mEq Na, per 24 hours). Note the profound fall in urine output for 28 hours after operation with a simultaneous rise in the specific gravity and chloride concentration. The fall in absolute chloride excretion in the urine is due to coincident impairment of salt excretion.

This relatively short-lived impairment of water excretion after operation was investigated in a different fashion by Dudley *et al.* (1954), who compared the effects of a rapid infusion of 600–800 ml. of 5 per cent. dextrose intravenously in normal subjects and in patients after operation. They showed that for some 36 hours after operation such an infusion did not give rise to an increased urine flow and diminished urine osmolarity as in normal subjects. Using the same technique, they investigated the effects of anæsthesia itself without operation, and showed that whilst one hour's pentothal anæsthesia caused no change in the rate of excretion of such a water load, three hours' ether anæsthesia after pentothal induction caused a distinct impairment of water excretion, which, however, returned to normal within 24 hours.

Salt Excretion

In 1936 Coller, Dick and Maddock clearly showed that the "surgically sick patient" was incapable of excreting salt normally, and that œdema could be produced relatively easily in such patients by giving an excess of salt. As emphasised previously (pp. 7–8), sodium is the ion of outstanding importance in the extracellular space, and

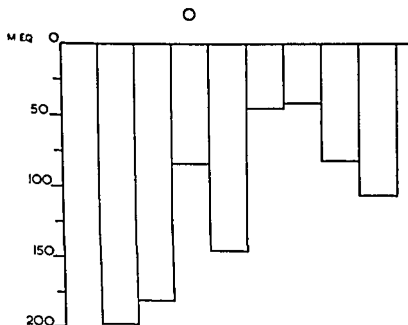


FIG 10.—Data from a case undergoing subtotal gastrectomy who received a constant intake of water and electrolytes throughout the period illustrated (see text for details). The graph shows the urinary sodium excretion in milli-equivalents each 24 hours. Following operation (O), this is diminished for 6 days, with the minimal excretion occurring on the third day. Note that this diminution in sodium excretion appears to occur in two phases.

although chloride excretion is similarly affected it is the impairment of sodium excretion which is of critical importance, and it is accordingly the sodium balance which gives the most significant information about this phenomenon.

Compared to that of water excretion, the impairment of salt (sodium) excretion is relatively prolonged, lasting for some 4–6 days after an operation of the severity of a partial gastrectomy. Figure 10 shows the 24-hourly urinary excretion of sodium of a patient before, during and after gastrectomy, who received throughout this period 4 litres of water and 160–170 mEq Na/24 hours, except on the day of operation itself, when there was an additional intake of 60 mEq Na contained in 0·5 litres of citrated blood. It can be seen that for some days after operation the urinary excretion of sodium fell sharply, and had not returned to normal at the conclusion of the balance study. This diminution in urine sodium excretion is not due to any excess abnormal loss, as from the alimentary tract, and in fact may lead to a distinct retention of this ion, which, as can be seen (Fig. 11), appears to occur in two distinct phases.

During the first 24 hours after operation impairment of salt excretion coincides with that of water excretion, with the result that there was during this period in the case illustrated (Fig. 11) a retention of both water and salt, as shown by the weight curve and sodium balance. As already shown, the impairment of water excretion on this day is independent of the salt intake, indicating that at this time the two phenomena are distinct and separable. On the 2nd–4th days after operation the sodium balance and weight curves show that there was a further impairment of excretion, with retention of both sodium and water. However, during this period the two are not separable. Figure 12 shows the data from a case given no salt for 4 days after operation and consequently in negative sodium balance throughout this time: it will be seen that after its initial rise the weight falls steadily, indicating that during this time water retention is secondary to and dependent upon sodium retention, or rather, that during this time the impairment of water and salt excretion are not separable, but are part of one phenomenon, the impairment of salt excretion being the primary event.

The difference between these two periods may be expressed by saying that immediately after operation there is a potential retention of salt and water, whilst later there is a potential retention of salt water. This difference is reflected in the type of urine secreted in the two phases. During the first phase, owing to the marked diminution of urine flow, the urine has a raised concentration of electrolytes, but in the second phase, the concentration is lowered. This contrast in the urine is well shown by the case illustrated in Fig. 11: on the days O and + 3 there was a marked impairment of sodium excretion

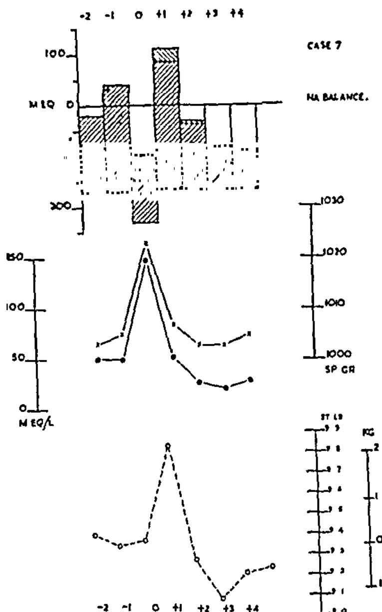


FIG. 11.—Complete sodium balance data from a patient undergoing subtotal gastrectomy, who received 4 litres of water each 24 hours. Following operation there is an impaired urinary sodium excretion, leading, on the intake given, to a definite retention of this ion. This appears to occur in two phases, both of which, as shown by the body weight, are accompanied, again on the intake given, by a retention of water. During the first 24 hours after operation the retention of sodium and water are independent, separable phenomena, but in the latter period the impairment of water excretion is dependent upon that of sodium. Note the difference in the urine electrolyte concentrations in these two periods.

with definite retention, and the absolute urinary excretion on each of these days was almost equal, yet the concentration of both sodium and chloride in the urine shows a fall of some 100 mEq/l. between the first and second of these days.

FIG. 12.—Data from a patient who received no salt after operation, except for that in 500 ml. of citrated blood on the day of operation The body weight on the morning after operation was not recorded, but thereafter there was a steady fall in weight until sodium was given, showing that during this period any water retention is dependent upon and secondary to a positive sodium balance.

In the cases illustrated in Figs. 10 and 11 the two periods of impaired salt excretion are clearly separated, but this pattern is far from invariable, and often the period of impaired salt excretion appears to be continuous (Fig. 13). Indeed, it is possible that the apparent separation of the two phases is an artefact due to the particular intake given to these patients. In the cases illustrated the balance studies were not continued long enough to observe the return to normality of salt excretion, but the evidence of other workers (Moore and Ball, 1952; Moore, 1953) suggests that such does not occur for some 4–6 days after an operation of moderate severity, that the duration of impaired salt excretion varies directly with the severity and length of the operation, and that, if salt retention occurs during this time, the retained salt and water are only excreted relatively slowly thereafter.

Potassium Excretion

Following operation there is an increased urinary excretion of potassium (Randall *et al.*, 1949; Moore and Ball, 1952), which is maximal on the day of operation, and is usually only of short duration, being over by the first or second post-operative day. This urinary potassium excretion is in relative excess of the nitrogen excretion, causing a rise in the urinary K:N ratio (see p. 78), and indicating that this urinary potassium loss cannot be due to tissue breakdown alone, but must be indicative of a mobilisation and excretion of intracellular potassium. If no potassium is given during the first few days after operation, this urinary excretion must lead to a negative potassium balance, but as far as is known the administration of potassium throughout the whole period will not prevent the development of a negative balance, at least during the first 24–48 hours after operation, though there does not appear to be any evidence of the effects of really high levels of potassium intake during and after the day of operation.

This potassium diuresis, and in particular its importance in relation to the development of potassium depletion, is discussed in greater detail in a later section. In that it leads to a negative potassium balance it is also, however, of particular importance in relation to the post-operative metabolic disturbance itself, as it has been shown by various workers (Black and Milne, 1952; Fourman and Ainley-Walker, 1952) that potassium deficiency can cause sodium retention. The balance studies so far illustrated here were performed on patients who received no potassium throughout the period of study, which started 4 days before operation. For comparison a further series of patients was studied receiving potassium throughout the period of observation (100 mEq K per 24 hours, save on the day of operation, when it was reduced to 50 mEq K).

The post-operative impairment of water excretion was not significantly affected by giving potassium (Fig. 14). Similarly the impairment of sodium excretion in the first 24 hours after operation was not affected. But in those patients receiving potassium the later impairment of sodium excretion was much less marked and less prolonged, so that in some cases the excretion of the retained salt and water was observed. Further, in these cases the separation of the impairment of salt (sodium) excretion into two distinct waves was more prominent. As would be expected, this lessened impairment of sodium excretion was reflected not only in the balances and weight curves, but also in the urine sodium concentrations, which did not fall to the low level seen in those cases receiving no potassium (Fig. 15). In view of the studies mentioned above, these effects of potassium administration are not surprising and there can be little doubt that potassium deprivation was partly responsible for the

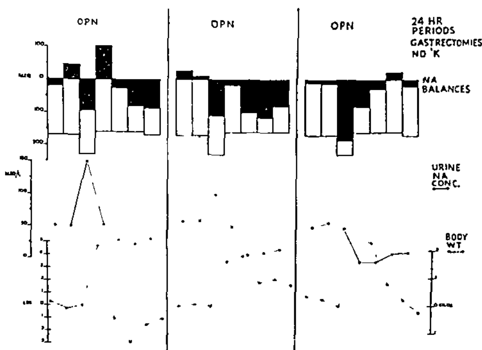


FIG. 13.—Sodium balance, urine sodium concentration and body weight changes in three patients undergoing partial gastrectomy. In all cases the weight is adjusted to zero on the morning of operation In the left-hand case the two phases of impaired sodium excretion are clearly separated, in the middle one they remain distinct, but in the third they have completely coalesced. The change in body weight and urine sodium concentration accurately reflect these differences in time between the two episodes of sodium concentration. In assessing the sodium balances it should be remembered that a normal subject on 100 gm. glucose daily loses nearly 200 mEq Na over 5 days (Gamble, 1951).

sodium retention noted by previous workers, though it is equally certain that it is not the main cause of post-operative impairment of sodium excretion.

Actually, the treatment in both these groups of patients (i.e. those receiving no potassium and those receiving it throughout) is artificial, in that the usual, present practice is for a patient to receive a normal potassium intake in his diet up to the day of operation, fol-

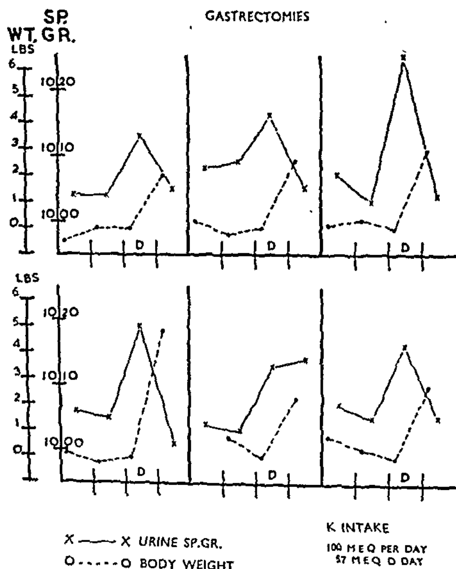


FIG. 14.—Body weight and urine specific gravity changes in six cases who received potassium throughout the period of study. These patients received 4 litres

lowed by no intake at all if he is receiving intravenous fluids. To mimic this state of affairs, 3 patients were studied who received potassium in their pre-operative feeds, but none on the day of operation or thereafter; the results were intermediate between the two main groups, but the later impairment of salt excretion tended to resemble that in the potassium-free group.

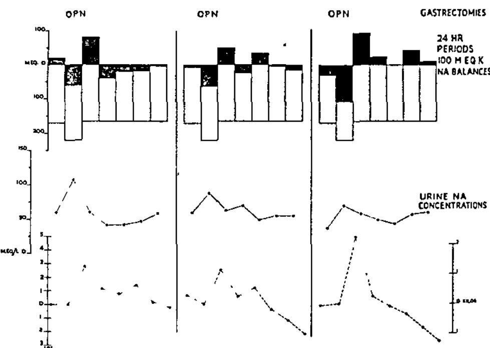


FIG 15.—Data from three cases undergoing partial gastrectomy, who received potassium throughout All three clearly show impairment of sodium excretion with retention on the day of operation, but the later impairment of excretion is less marked (*cf* Fig 13).

Nitrogen Excretion

After operation the urinary excretion of nitrogen is increased, this increase lasting for 24 hours after an operation of the magnitude of a sub-total gastrectomy, but somewhat longer after larger procedures. Regardless of the size and type of the operation, the maintenance of an adequate nitrogen (protein) intake will not apparently prevent this urinary nitrogen excretion from leading to a negative balance, but it is to be noted that in all observations on this point the calorie intake was inadequate, even in those cases given adequate nitrogen. The increased nitrogen excretion in the urine is indicative of an increased breakdown of body tissue, but it must be stressed that this period of apparently obligatory tissue breakdown and negative nitrogen balance is relatively short-lived. Many patients

are in negative balance for many days after operation, but save for the first 2–3 days this is an expression of an inadequate protein and calorie intake, and must be clearly distinguished from the early, inevitable loss. (See later section for fuller discussion of these points.)

Plasma Electrolyte Concentrations

The post-operative metabolic response is reflected not only in alterations in the renal excretion of water, electrolytes and nitrogen, but also in changes in the concentration of electrolytes in the plasma. These changes consist of a fall in the plasma sodium and chloride figures, and a rise in the potassium concentration. Regardless, within limits, of the intake of water and salt, major surgical procedures are regularly followed by a fall in the concentration of sodium and chloride in the plasma. Zimmerman (1951) observed and commented on this extracellular dilution, and it has been recorded by many workers since. Moore (1954) points out that this fall is a normal response to injury and that the plasma sodium concentration may fall as low as 125 mEq/l. This fall in plasma sodium concentration, which is accompanied by a proportionate fall in the chloride concentration, takes 2–3 days to reach a maximum, but the figures may remain low for up to a week after operation. In many cases this fall may be in part due to a retention of water, as in the patients observed by ourselves (Le Quesne and Lewis, 1953), who showed a marked fall in plasma sodium, chloride and protein concentration in the first 24 hours after operation. However, there is no doubt that this fall also occurs in patients who do not receive an intake allowing of any significant water retention, and that essentially it is due to a shift of sodium and chloride into the intracellular space (see Zimmerman, Casey and Bloch, 1956), a shift which appears to be an intrinsic part of the metabolic response to surgery.

Coinciding with this fall in the plasma sodium and chloride concentrations there is a rise in the potassium, which may reach a level of 4·7–5·2 mEq/l. This rise is indicative of a mobilisation of intra-[illegible] ...ical, this rise in the plasma potassium concentration and fall in that of sodium and chloride are especially pronounced after the operation of mitral valvotomy (Wilson *et al.*, 1954), and Bruce *et al.* (1955) bring forward evidence suggesting that this exaggerated response is a result of the patient's abnormal cardio-circulatory state.

ÆTIOLOGY OF THE METABOLIC RESPONSE

In recent years it has become apparent that the metabolic response to trauma is associated with an increased secretion of adrenocortical hormones, and in general it is considered that this adrenocortical

lowed by no intake at all if he is receiving intravenous fluids. To mimic this state of affairs, 3 patients were studied who received potassium in their pre-operative feeds, but none on the day of operation or thereafter; the results were intermediate between the two main groups, but the later impairment of salt excretion tended to resemble that in the potassium-free group.

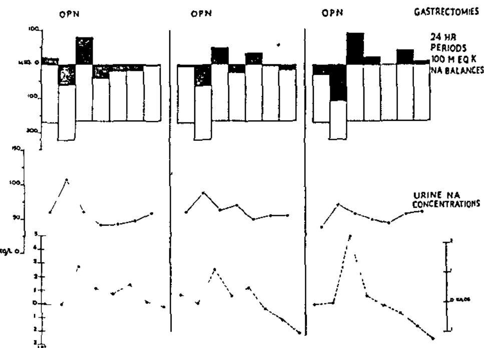


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Moore *et al.* (1955) showed that operation is not followed by a significant rise in the urinary excretion of 17-ketosteroids, but that there is a constant rise in the urinary excretion of hydroxycorticoids, lasting for 1–5 days, and they found that there was a close correlation between the magnitude of the operation, the increase of 17-hydroxy-

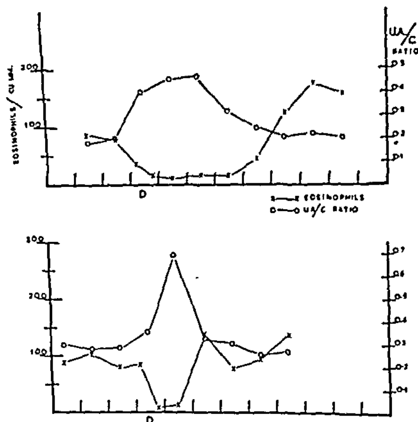


FIG. 16.—Graphs showing the eosinophil count and urinary uric acid/creatinine ratio in two cases following partial gastrectomy. Immediately after operation there is a sharp fall in circulating eosinophils and rise in the uric acid/creatinine ratio, but the return of these indices to normal is variable, and, in the cases observed, bore no constant relationship to the sodium balances.

corticoids in the urine and the duration of negative nitrogen balance, but there appeared to be no correlation between these and the impairment of sodium excretion. Jepson *et al.* (1956), estimating not the 17-hydroxycorticoids but an essentially similar group the 17-ketogenic steroids, found that their urinary excretion also rose in a group of patients following partial gastrectomy, the rise being maximal in the first 24 hours and lasting 2–4 days.

It has also been clearly established that not only the urinary excretion but also the plasma level of 17-hydroxycorticoids rises after

secretion is responsible, in large part, for the response. However, it is also apparent that factors other than a simple increase in adrenal secretion are involved, of which the most important appear to be a modificatioh in the response to the adrenal hormones, other hormonal factors, in particular the antidiuretic hormone of the neurohypophysis, and purely hæmodynamic factors. In considering these various factors, it must be borne in mind that in the great majority of patients these changes take place at a time when their intake of protein and calories is grossly impaired, but it is certain that this impairment of intake is not in itself responsible for the changes that occur.

Increased Adrenocortical Secretion

It is well established that the administration of either ACTH or cortisone causes a retention of sodium and water, together with an increased excretion of nitrogen and potassium, and it is clear that the general pattern of the metabolic response to operation closely simulates the results of administering either of these substances. Furthermore, these metabolic responses are accompanied by other changes highly characteristic of a release of adrenocortical hormones. The administration of ACTH to a patient with normal adrenal glands regularly causes a fall in the level of circulating eosinophils (Roche, Hills and Thorn, 1950), and, as shown by Roche, Thorn and Hills (1950), operation is followed within a few hours by a similar fall. This fall in circulating eosinophils is characteristically accompanied by similar but inverse changes in the urinary uric acid/creatinine level, and these changes in two typical cases are illustrated in Fig 16. The rate of return of both these indices to normal appears to be very variable, and to bear no constant relationship to any of the major metabolic disturbances, in particular the impairment of sodium excretion, but it may be related to the secretion of certain adrenocortical hormones (Steenburg *et al.*, 1956). Frawley and Thorn (1951) have also shown that the administration of ACTH causes a fall in the salivary Na:K ratio, and this again has been shown to occur after operation (Tobiassen *et al.*, 1954). Further, the sweat sodium concentration falls after operation (Johnson *et al.*, 1950), a change known to result from the action of salt-retaining corticoids. The occurrence of all these phenomena simultaneously with the metabolic change already described is strong evidence that both are in fact due to the same cause.

But of greater significance than these facts is the rapidly accumulating evidence of increased adrenal hormone production after a surgical operation. As far back as 1944 Venning *et al.* showed that after operation there is an increased urinary excretion of cortin-like substances, and in recent years more specific studies have been made.

stress. Campbell *et al.* (1954) measured the urinary nitrogen excretion in rats following fracture of a long bone, and found that in adrenalectomised rats fed a fixed, daily dose of cortisone, such a fracture was followed by a nitrogen diuresis similar to that in normal, control animals, suggesting that an increased output of adrenal hormones is not essential for the metabolic response to trauma.

More recently metabolic studies on patients undergoing total adrenalectomy have been interpreted as supporting this "permissive" theory. Stuart Mason (1955) made observations on patients undergoing this operation for carcinoma of the breast, who received a fixed dose of cortisone, usually 80 mgm. for 2–4 days before operation and thereafter, and found that, as judged by their sodium chloride, potassium and nitrogen excretion, all showed an essentially normal response to surgery. Assuming that the pre-operative cortisone would have suppressed endogenous corticoid excretion, he argues that these results suggest that in the normal subject the post-operative response is not directly related to the level of circulating corticoid, but rather that there are certain biochemical responses to trauma which will function provided there is a constant supply of hormone.

It may well be, however, that these results are due to the slower inactivation of corticoids after trauma, so that any given dose, of exogenous or endogenous origin, has a more prolonged and pronounced effect. Steenburg and Gangong (1955) in animals, and Sandberg *et al.* (1954) in humans, have shown that following operation the metabolism of intravenously administered hydrocortisone is impaired. Further Steenburg *et al.* (1956) have shown that ACTH administered 2–8 days after operation causes a higher rise in the plasma level of 17-hydroxycorticoids than a similar dose given before operation, and that the normal post-operative rise in plasma corticoids is higher than that following an injection of 25 mgm. of ACTH, which they believe to cause maximal adrenal stimulation. On the basis of these results they believe that after operation there is not only an increased excretion of 17-hydroxycorticoids, but also an impairment of their rate of conjugation and destruction, the rise in plasma levels resulting from a combination of these factors. Similar conclusions were reached by Tyler *et al.* (1954), who measured the plasma 17-hydroxycorticoid level after operation and also the hepatic clearance of bromsulphalein. They found that in general the magnitude of plasma corticoid rise correlated with the degree of impairment of bromsulphalein excretion and the length and severity of the operation, and they suggested that impairment of their removal by the liver was one of the factors in the post-operative rise in the plasma level of corticoids.

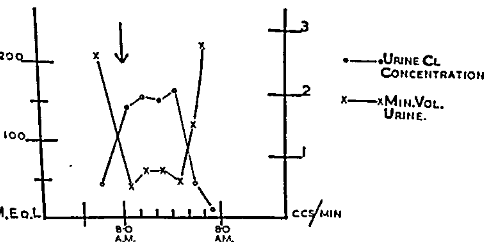
operation. Sandberg *et al.* (1954) found the plasma level of these corticoids to be raised after the induction of anæsthesia, during and after surgery. Elman *et al.* (1955), from studies on 24 adults undergoing elective surgical procedures, reported a rise in the plasma level of 17-hydroxycorticoids, maximal 8–12 hours after operation and returning to normal usually by 24 hours. Steenburg *et al.* (1956) reported similar findings: in general they found that the levels rose from 5–15 γ per cent. pre-operatively to 50–70 γ per cent. immediately after operation, and returned to normal again within 48 hours; but they found no constant relationship between the time at which the plasma level and urinary excretion of 17-hydroxycorticoids returned to normal.

The 17-hydroxycorticoids are essentially glucocorticoids, that is to say they influence mainly carbohydrate and protein metabolism, and their influence on electrolyte exchanges is less marked. However, there is also evidence that after operation there is an increased excretion of the most active mineralocorticoid, aldosterone. Using a bio-assay method, Llaurado (1955a) showed that there is a sodium-retaining, potassium-excreting factor in the urine after operation, and adduced strong evidence that this is in fact aldosterone. He found that the levels of this substance excreted correlated with the urinary Na/K ratio before and after operation. In a later paper he (Llaurado, 1955b) showed that the amount of aldosterone excreted in the first 24 hours after operation is as much as 8 times that excreted before operation. Zimmerman *et al.* (1956) have also reported finding increased amounts of aldosterone in the urine in the first 24 hours after operation, but so far no studies have been published on plasma levels of this hormone after operation.

Modification in the Response to Adrenocortical Hormones

Whilst there can be no doubt that operation is followed by an increased output of adrenocortical hormone, it has been suggested by some workers that this output is not in fact the cause of the metabolic response, but rather that, as a result of trauma, the tissues require more adrenal steroids and the increased secretion of hormones is in response to this demand. This is the theory of the so-called "permissive" role of the adrenocortical hormones, in that it postulates that these hormones are required to allow the tissues to respond to trauma, without themselves actually causing the response. This "permissive" theory, propounded by D. J. Ingle, was originally based on animal experimental work. Thus Ingle and Nezamis (1950), observing the effect of stress upon the glycosuria of adrenalectomised-depancreatised rats, interpreted their results as showing an increased utilisation by the tissues of cortical hormones following

300
200
100
URINE CL
MM %
0
5 MU
PITRESSIN
50
100
MINS
10
8
6
4
2
CC/MIN
0



comparison, the upper graph shows the effect on urine volume and chloride concentration of an injection of pitressin (slightly modified from Lewis, 1953). Note that both graphs show similar changes, save that after operation these are much more prolonged.

The Antidiuretic Factor

All the early and most of the later work on the metabolic response to trauma has centred round the problem of salt retention following operation. However, in 1949 Cooper, Iob and Coller showed clearly that there was a retention of water after operation, with a diminished flow of urine of high specific gravity. This was also shown by Moyer *et al.* (1949) and by Ariel (1951), the latter recording the resultant weight gain. However, none of these workers defined the duration of this water retention in relation to that of sodium, or described the part it plays in the whole post-operative disturbance. Our own results (Le Quesne and Lewis, 1953) show clearly that the impairment of water excretion is independent of sodium administration or retention, begins at the start of the operation and lasts for some 24–48 hours thereafter. These findings were confirmed by the work of Dudley *et al.* (1954).

Noting that the post-operative oliguria occurred at the same time as the fall in circulating eosinophils, Hardy (1950) suggested that the two were related, and that the oliguria was due to a secretion of adrenocortical hormones. However, there is no reason to believe that these two events are causally related and, indeed, the features of the impairment of water excretion (oliguria, raised urine specific gravity, raised electrolyte concentration) in no way resemble those of any known adrenocortical hormone.

These features are in fact identical with the effects of secretion of the antidiuretic hormone of the posterior pituitary gland. Figure 17 shows a comparison between the effects of an injection of Pitressin and those of an operation on the rate of urine flow and urine specific gravity: it will be seen that in both instances the changes are precisely similar, except that after operation they are greatly prolonged. Dudley *et al.* (1954) also noted this similarity, and observed that after operation there is a close correlation between urine flow rate and total solute excretion, a feature characteristic of maximal renal tubular absorption of water due either to endogenous or exogenous Pitressin. Investigating this point from a different angle, Eisen and Lewis (1954) carried out a bio-assay of the urine passed in the first 24 hours after operation by 18 different patients, and found it to contain an antidiuretic activity similar to that found in the urine of normal subjects after the injection of Pitressin or stimulation of the supraoptico-hypophysial system by nicotine. They put forward strong reasons for ascribing this action to the presence of antidiuretic hormone in the urine, and it seems most probable that this hormone is in fact responsible for the phase of impaired water excretion occurring immediately after operation. Further strength is given to the belief by the observations of Hayes and Coller (1952), who studied the post-operative urine output in a patient with diabetes insipidus

retention. By causing alterations in volume of the body compartments the intake the patient received will to some extent modify the pattern of response, as will the occurrence of any circulatory collapse during or after operation, but these modifications will usually only be minor superimpositions on the basic pattern dictated by the factors discussed, of which the outstanding are undoubtedly an increase in adrenocortical and antidiuretic activity.

The Stimulus to the Metabolic Response

It remains to consider in what way an operation, or any trauma, leads to these various results. With respect to the secretion of adrenocortical hormones, it appears that nervous impulses arising in the injured area and passing to the hypothalamus may be the main afferent stimuli. Hume (1953) describes the portal venous system connecting the hypothalamus with the pituitary, and showed that, in animals, destruction of this system or certain portions of the hypothalamus abolished the metabolic response to trauma, as measured by the eosinophil response. Further, he showed that electrical stimulation of the hypothalamus caused a typical response, but moderate trauma to a denervated extremity caused no metabolic response. On the basis of these observations Hume puts forward the hypothesis that nerve impulses arising in the injured tissue stimulate the hypothalamus, causing release of an humoral agent which is carried in the portal system to the pituitary, causing a release of ACTH and so a secretion of adrenocortical hormones. There must, however, be other mechanisms involved as well, for, as pointed out by Hume (1953), severe trauma to a denervated limb will cause a modified response, which almost certainly plays a part in the metabolic response. These other factors remain unknown, but it seems likely that the circulatory effects of trauma by stimulating stretch receptors (see earlier section) and baroreceptors may well be important afferent stimuli.

The normal physiological stimulus to the secretion of antidiuretic hormone is a rise in plasma osmotic pressure (Verney, 1946), and it is possible that this might occur during operation, when insensible loss is often increased and is either not replaced or is replaced by saline. However, in one of our cases care was taken to give 5 per cent. glucose intravenously during operation at such a rate as to preclude this happening, and estimation of plasma sodium and chloride before, during and after operation confirmed that no hæmoconcentration occurred. But the impairment of water excretion in this case was no less marked than in those cases in which no such precautions were taken. So whilst such hypertonicity may contribute towards the release of the antidiuretic hormone, it is clearly not the main cause after operation.

and panhypopituitarism. Despite the administration of adrenal cortical hormones during and after operation, they found that the oliguria during the first 6 hours post-operatively was not so marked as in a normal subject.

Hæmodynamic factors

Quite apart from these endocrine factors, it appears that during and immediately after operation there are renal hæmodynamic changes which probably play a definite role in the metabolic response. Coller *et al.* (1943) showed that the glomerular filtration rate falls during anæsthesia. Further, Lee *et al.* (1953) showed that anæsthesia causes a fall in renal blood-flow, and Leaf *et al.* (1954) found that, in dogs, this is accompanied by a reduction in urine flow and total solute excretion. The glomerular filtration rate has been shown to fall not only under anæsthesia but also during an operation (Habif *et al.*, 1951), though Ariel and Miller (1950) found that the rate rapidly returned to normal after operation. Further, it is to be borne in mind that during and immediately after operation there may well be significant changes in the filling of the heart and great thoracic veins, as a result of circulatory changes due to hæmorrhage and shock and the type of anæsthesia used. Such changes are capable of causing distinct alterations in urine flow (see pp. 14–15) and may well influence the metabolic response. There is no reason to believe that these hæmodynamic changes play a major role in the metabolic disturbance, but they may play a significant part in the changes occurring during, and in the first few hours after, operation.

Summarising these considerations, it may be concluded that the entire metabolic response to operation is due to an interaction between these factors, and possibly others as yet unrecognised. The essential background to the response appears to be an increase in adrenocortical activity, due in part to an increased secretion of hormones by the glands and in part to an impairment of the rate of breakdown and inactivation of these hormones. During the first 24–48 hours after operation there is also a secretion of antidiuretic hormone, and in addition hæmodynamic factors play a part. In any given case the exact pattern of response depends upon the relative intensity and interaction of these various factors, and considering their complexity it is not surprising that there are differences in the response from patient to patient. It is tempting to believe that the apparently biphasic nature of the impairment of sodium excretion is due to aldosterone and 17-hydroxycorticoids affecting the kidney at different times. But there is no evidence in favour of this, and it may well be that the break in the impairment of sodium excretion is to an extent artefactual, and dependent upon the intensity of the impairment of water excretion and the amount, if any, of water

ADMINISTRATION OF FLUID AND ELECTROLYTES IN THE UNCOMPLICATED CASE

PRE-OPERATIVE ASSESSMENT

The great majority of patients undergoing surgical operations are on a normal diet until a few hours before operation, and are therefore in normal electrolyte and water balance. However, many patients are operated upon for conditions commonly giving rise to fluid imbalance, and in such cases, even though abnormal losses or deficient intake may not be obvious, it is important to make sure that the patient is in normal balance before operation. Post-operative morbidity and mortality are significantly higher in patients submitted to surgery whilst in fluid and electrolyte imbalance, and further the correction of fluid imbalance is more difficult after than before operation, owing to the metabolic changes then in progress. For both these reasons, a careful pre-operative assessment of the state of hydration is essential in any patient potentially in imbalance. When vomiting or thirst is pronounced, the problem is obvious, but where the manifestations are less overt it is easy to overlook a quite severe, especially a long-standing, deficiency of water and electrolytes, with raised blood urea and other serious sequelæ. It is in these cases particularly that simple pre-operative precautions can save serious post-operative complications.

This pre-operative assessment, when necessary, need not involve any complex biochemical investigations. Its most important feature is a careful clinical scrutiny of the patient, with particular reference to mental state, blood pressure, condition of the skin and subcutaneous tissues, condition of the mouth, and the tone of the muscles. In association with this, the urine volume should be noted, together with its specific gravity and chloride concentration. Of particular value is a simple recording of the fluid intake and urine output over 2–3 days, when a persistently low urine output, particularly in association with a good fluid intake, will clearly indicate a deficiency. These simple observations, together with an estimation of the blood urea, are sufficient to rule out any significant degree of depletion, and there is no necessity to perform plasma electrolyte estimations except under exceptional circumstances. If the blood urea remains raised despite a consistently good urine output, it is, of course, essential to investigate renal function, and probably the water concentration and dilution test gives as useful information as any other

In 1949, Burnett *et al.* suggested that anæsthesia caused a release of antidiuretic hormone, and Ariel and Miller (1950) thought that such a release might occur during abdominal operations. Further, Habif *et al.* (1951) suggested that the reduction in urine flow following the injection of morphine or pethidine was due to a release of this hormone, and Moyer *et al.* (1949) suggested that all these factors play a part in the post-operative oliguria. Lewis *et al.* (1952) have produced evidence that emotional and other stimuli may be potentiated by morphine to produce a prolonged antidiuretic release. It seems probable that a summation of all these factors—namely emotion, morphine, pethidine, anæsthesia and trauma, with, possibly, temporary hypertonicity—is the effective stimulus to the release of antidiuretic hormone on the day of operation.

SUMMARY

All surgical operations, and similarly other forms of trauma, such as burns and fractures, provoke and are followed by a complex metabolic disturbance, which in general varies in magnitude and duration directly with the severity of the operation or trauma. The main features of this disturbance are (1) An impairment of water excretion, lasting some 24–36 hours after operation, and characterised by a low urine output with raised specific gravity; (2) An impairment of sodium excretion lasting 3–6 days after operation and characterised, after the first 24 hours, by a marked lowering of the urine concentration of sodium and chloride; (3) An increased urinary excretion of potassium, lasting some 24–48 hours after operation, and indicative of a mobilisation of intracellular potassium; (4) An increased urinary excretion of nitrogen, indicating an increased tissue breakdown, and lasting a variable time; (5) A fall in the plasma sodium and chloride concentrations and a rise in the plasma potassium concentration, all three usually being maximal 2–3 days after operation.

The main cause of this metabolic response is an increase in adrenocortical activity, due in part to an increased secretion of adrenocortical hormones and in part to an impairment of their rate of destruction. In addition there is almost certainly a secretion of antidiuretic hormone. Further, various hæmodynamic factors and the actual intake given influence the response, and in any case the detailed pattern of response depends upon the interaction of these various factors.

cause any dangerous retention. But when fluids are given parenterally this automatic safeguard of the patient's own desires is no longer effective, and must be replaced by the conscious control by the surgeon of the amount of fluid given. Further, in general, intravenous fluids are only required after major operations, and as already stated the magnitude of the post-operative changes depends directly on the severity of the operation, so that it is in just those cases requiring intravenous fluids that the risks of overloading are greatest.

PRACTICAL APPLICATION OF THE POST-OPERATIVE METABOLIC CHANGES

In normal health the kidneys have an enormous flexibility of function, and can readily excrete from the body any excess water and salt. The essential result of the post-operative metabolic changes is that after operation this characteristic flexibility is temporarily replaced by a comparative rigidity of function, so that the kidneys are unable to excrete excess water and salt, which must then necessarily be retained in the body. In the first 24–48 hours after operation this rigidity of function affects the kidneys' ability to excrete both salt and water, but after the impairment of water excretion has worn off only salt excretion is affected. Accordingly, after operation there is not only a distinct danger of overloading a patient with saline, but during the immediate post-operative period there is a similar danger of overloading with glucose solutions, the form in which water is usually given post-operatively. In fact, these risks are so real that it is possible to produce recognisable clinical syndromes by excessive administration of either water or salt after operation.

The dangers of giving excess quantities of saline after an operation have been clearly recognised for many years, and in their paper describing post-operative salt intolerance Coller and his associates (1945) described 5 cases made œdematous in this way. The similar danger attendant upon the excessive administration of water (i.e. glucose solutions) has not been so clearly recognised, though in fact during the first 48 hours after operation the risk is very real and water intoxication can be caused, this post-operative complication being first described by Helwig, Schutz and Curry in 1935. Both these conditions are described fully in a later section (pp. 93–98).

These conditions of water intoxication and salt retention with œdema formation are, of course, gross manifestations of the body's inability to excrete water and salt normally after operation, and as such are not commonly seen. Less severe examples of overloading undoubtedly occur much more frequently, and though they cause less dramatic signs and symptoms, these can none the less be both unpleasant and dangerous to the patient. Thus, undue water retention increases post-operative nausea and vomiting, and probably

(see Appendix B, Case1). It may well be necessary to operate upon a patient with poor renal function, but it is important to know this before operation, so that wrong conclusions will not be drawn if, for instance, a markedly raised blood urea is found a few days after operation.

GENERAL CONSIDERATIONS

In most cases not only is the patient on a normal intake until a few hours before operation, but is able to return to a similar intake within a few hours after operation. Provided no post-operative complications occur, such cases present no problem with regard to fluid balance, and the intake can well be left to the desires of the patient controlled by the discretion of the nursing staff. After all operations of any severity it is wise to keep a simple record of fluid intake and output for 1–3 days after operation, so that serious deficiencies or abnormalities in intake or output may be noticed early. Provided that after 24 hours post-operatively the urine output exceeds 1 litre (40 oz. approx.) per 24 hours, and there are no abnormal losses, it can be safely assumed that all is well. If the urine output is less than this, it is wise to consider the intake figures and *decide whether the low urine output is due to deficient intake*, abnormal losses, or some other cause such as renal damage, so that appropriate treatment can be started in good time (see p. 53 for fuller consideration of this problem). But in the large majority of routine surgical cases such problems will not arise, and the metabolic changes described in the previous section demand no specific consideration.

However, in cases in which fluid and electrolytes have to be given intravenously or by proctoclysis more serious attention must be paid to the amounts administered. It must be emphasised straight away that the intravenous route is not a simple alternative to oral feeding; the administration of intravenous fluids carries slight but definite risks, has certain complications and imposes increased responsibilities on the surgeon in charge of a case. Judiciously controlled, intravenous fluids constitute one of the most valuable advances in surgery, but their abuse can be highly dangerous to the recipient, especially in the few days immediately following an operation, and fluid administration by mouth is always preferable when possible. During the post-operative period the essential risk associated with the intravenous administration of fluids is that of overloading the patient with water and/or salt, and herein lies the main practical significance of the changes described in the previous chapter. These changes occur, of course, after all operations, but when a patient can take fluid by mouth it is in the highest degree improbable that he will, unless unwisely forced, take sufficient to

put are frequent, owing to increase in the cutaneous and respiratory loss, and to some pooling in the intestines from mild, transient ileus. Unless the intake is designed to allow some margin for these occurrences, there will be a constant danger of the urine *output falling to* undesirably low levels, and frequent additions to the intake will be necessary. Accordingly, in planning the basic intake a balance must be struck between the dangers of overloading and the disadvantages of an intake which allows for no reasonable variation in output.

In calculating the water requirements, the first consideration must be replacement of the insensible loss. In the average case this will amount to about 1000 ml. per 24 hours, but it may well exceed this figure. In addition, adequate water should be given to allow of a reasonable urine output, which must exceed 500 ml. per day if nitrogen retention is to be prevented. But many surgical patients have some diminution in renal concentrating power, and after operation there is an increased load of metabolites for excretion, so that an output of 1·0–1·5 litres per 24 hours is desirable, thus giving a total required intake of 2·5–3·0 litres per 24 hours. Bearing in mind the considerations set out above, 3 litres of water per 24 hours appears to be the optimal intake, with the important exception that during the first 24 hours after operation this should be reduced to 2 litres, owing to the marked tendency to retain water during this period. There is no reason to believe that such an intake is sufficiently high to cause dangerous overloading provided certain simple precautions are taken (*vide infra*), particularly when it is recalled that in the series of special study cases (see previous chapter) the daily intake was 4 litres, without apparent harm. It may well be that on an intake of 3 litres per day some patients pass approximately 2 litres of urine per day, but this is not a sufficiently high volume to cause inconvenience, and in many cases—e.g. prostatectomy, ureteric transplantation, etc.—a high rate of urine flow is desirable.

With respect to the salt intake, a consideration of the events in the immediate post-operative period suggests that a complete withholding of salt is illogical. The outstanding metabolic disturbance during the first 48 hours after operation is an impairment of water excretion; this must cause a tendency to hypotonicity, which can only be accentuated by giving no salt, Further, during subsequent days there is necessarily a small salt loss in the sweat, in the urine and in many cases, for reasons mentioned above, into the gut. No doubt during this period 2 gm. of salt per day would suffice, but on grounds similar to those relating to the water intake, 4·5 gm (80 mEq) of salt per day seems preferable, and there is no reason to believe that this is an intake sufficient to cause harmful retention. Furthermore, this figure has the additional practical advantage of being that amount contained in one ½-litre (pint) bottle of normal saline.

contributes to the mental depression and fatigue seen after operation. Salt retention, though not of a degree to cause clinically obvious œdema, can easily cause œdema in wounds, at intestinal suture lines and at the lung bases, leading to a whole series of complications which are likely to delay the patient's recovery. Accordingly, during the post-operative period of comparative rigidity of kidney function, it is important to specify and administer a basic water and salt intake which, when given intravenously, is not sufficiently large to allow of any dangerous degree of retention and overloading, yet which is adequate to the body's requirements, provided there are no abnormal losses, as by vomiting, fistula formation, etc.

BASIC INTAKE IN THE POST-OPERATIVE PERIOD

Bearing in mind that during the first 48 hours after operations the main danger is that of overloading with water (i.e. glucose solutions) and that thereafter this danger is confined to saline, it would ideally be best to give an intake which varied from day to day, in accordance with the changes in the metabolic disturbance. To design such an intake would be comparatively simple, but experience shows that in practice such a scheme is too complex and leads to confusion rather than clarity. If at all possible, it is desirable to specify and administer a relatively fixed intake, which need only be varied under exceptional circumstances (*vide infra*).

Although most workers' recommendations for this basic intake are not grossly at variance, there is no generally accepted level of intake for the critical post-operative days. In 1944 Coller *et al.*, emphasising the danger of its excess administration, recommended that no saline should be given on the day of operation and during the two subsequent days, during which time only 5 per cent. glucose should be given intravenously. Discussing the same subject in 1949, Coller and de Weese stressed the importance of the earliest possible return to oral fluids, recommended that no saline should be given *intravenously during the 48 hours post-operatively and that thereafter* it should only be given to replace abnormal losses. In the same year Elman *et al.* (1949) recommended that the basic intravenous intake after operation should consist of 2 litres of glucose per 24 hours, containing 2–4 gm. of a mixture of sodium and potassium chloride. Somewhat modifying this intake, in 1952 Elman and Weichselbaum suggested that the 2 litres of fluid should contain Na 34 mEq, K 33 mEq, Cl 34 mEq and P 18 mEq.

A critical consideration of these various recommendations suggests that they are probably over-influenced by the fear of excessive administration, and represent minimal rather than optimal figures. Following operation, comparatively minor increases in out-

(2) No potassium should be given in the first 24 hours after operation.

In view of the fact that both these two modifications are dictated by the marked impairment of water excretion which occurs after operation, it is perhaps more logical and elegant to increase the intake and add potassium not arbitrarily at the end of the first 24 hours, but at the time when water excretion returns to normal. This occurs, in the great majority of cases, some time in the second 24-hour period post-operatively, and is clearly marked by a distinct rise in urine volume and fall in specific gravity. We have treated a large number of patients by each method, with no detectable difference in results. The former method is the simpler, but the latter is probably preferable and is now our standard practice.

It must be stressed that with an intake as specified, deliberately designed slightly on the generous side, these amounts should not be exceeded unless there is clear evidence of abnormal losses.* Further, the intake described should be regarded not as a rigid schedule, but rather as a basic guide, and it must always be remembered that the main danger in the post-operative period is that of overloading the patient, not of giving too little. Accordingly, in any case of doubt it is wise to restrict rather than increase the intake. Thus, if there is evidence of unusually prolonged impairment of water excretion, or in very small or frail patients, no harm can come from omitting 0·5–1·0 litres of the water intake, unless there are evident abnormal losses. (See later section for replacement of abnormal losses.)

METHOD OF ADMINISTRATION

This intake of 3 litres per day has the distinct practical advantage that it entails the patient receiving every 12 hours three of the standard 500-ml. bottles of intravenous fluid. On the day of operation itself, when the water intake is restricted to 2 litres and no potassium is given, the intake is provided quite simply by three bottles (1·5 litres) of 5 per cent. or 10 per cent. dextrose and one bottle (0·5 litre) of normal saline, containing 4·5 gm. NaCl. The requisite quantity of salt can equally well be provided by the appropriate quantities of one-third or one-fifth N saline.† But there is no evidence that by giving the saline in a hypotonic form post-operative retention is affected, and on more general grounds this practice is to be deprecated. The conscious, exact control of the salt intake is so important that any

* It must be borne in mind that the actual water available to the body is [illegible]

[illegible] these solutions are sufficiently hypotonic to cause hæmolysis, hæmoglobinuria and kidney damage if administered at all fast.

In addition to this 3·0 litres of water and 4·5 gm. (80 mEq) of salt, it is becoming clear that the basic intravenous intake should contain potassium salts. For the whole period that a patient is on intravenous fluids consisting of glucose and saline there is a steadily mounting negative potassium balance which is augmented by the potassium diuresis occurring after operation. This continuing potassium loss in the absence of intake is one of the main causes of post-operative potassium deficiency states, and it is illogical to let this condition develop when in most cases it can be quite simply prevented. Further, as shown previously, potassium deficiency accentuates the impairment of sodium excretion, and as a corollary to this, in cases receiving potassium the urinary sodium and chloride concentrations do not fall so markedly after operation (see Figs. 14 and 16).

For these reasons it is clear that potassium should be given as a routine measure, but the desirable intake is far from clear. Our own results show that after operation 100 mEq K can be given daily with impunity, and that 50 mEq on the day of operation is apparently harmless, although an intake of this amount is probably too high. Winfield *et al.* (1951) used very much smaller amounts of potassium, together with minimal amounts of magnesium, calcium and acetate, and claimed that this diminished sodium retention, though in discussing Howard and Mason's paper (1946), Reifenstein suggested that 50 mEq per day might not be adequate for this purpose. In a more recent paper, Lans, Stein and Meyer (1952) report that they have seen the development of hypokalæmia on an intake of 55 mEq K per day, and indeed it is probable that in the face of copious alimentary losses some degree of potassium deficiency cannot be prevented. From a consideration of these facts and in the light of our present knowledge, a reasonable daily intake of potassium seems to be 80 mEq K—that is, the amount in 6·0 gm. KCl. We have now given this amount of potassium as the standard daily intake to a large series of patients on intravenous fluids, both before and after operation, with no adverse results, and have found that it does, in fact, largely prevent the development of post-operative potassium deficiency. However, in view of the diminished urine flow immediately following operation, it is probably wise not to give any potassium post-operatively for the first 24 hours, and there is no evidence that such a short period of absent intake has harmful effects.

Summarising these considerations, it can be said that the basic post-operative intravenous intake each 24 hours should consist of 3 litres of water, 4·5 gm. (80 mEq) of NaCl and 6·0 gm. (80 mEq) KCl, with two important modifications.

(1) During the first 24 hours after operation the water intake should not exceed 2 litres.

(2) During the first 3–4 days after operation a markedly lowered urine chloride (or sodium) concentration does *not* necessarily indicate salt depletion: it is probably a manifestation of the impaired ability to excrete salt, and should certainly not *per se* be taken as an indication for increasing the salt intake.

(3) Normally the urine output varies directly with the water intake; following operation this is not true. Accordingly, marked variations in the urine volume with a fixed intake are not *per se* indications for increasing or decreasing the intake.

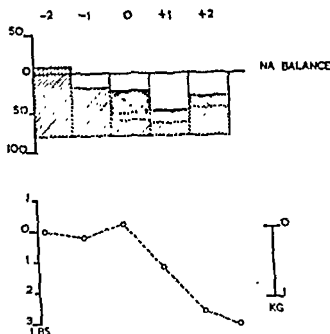


Fig. 18.—Sodium balance and weight curve from a patient undergoing gastrectomy, who received the standard intake recommended in the text. Note the steady fall in weight after operation and insignificant sodium retention.

(4) Following operation there is regularly a fall in the plasma sodium and chloride concentrations, and a rise in the potassium concentration (p. 33). These changes are expressions of the metabolic response to surgery, and must not be construed *per se* as indications to alter treatment.

In view of these marked alterations in urine volume, gravity and chloride concentration, the most reliable checks on fluid balance during the post-operative period are provided by the clinical condition of the patient, together with accurate measurement and recording of intake and losses by all routes. Experience shows that with these simple observations any serious disturbance in fluid balance can be quickly detected, but there is no doubt that the control and

technique which minimises this control is undesirable. Practical experience shows that, in fact, confusion and mistakes are more likely to occur when using dilute saline solutions, whereas if isotonic saline is used the daily ration is provided by one bottle, and thereafter only glucose is given.

On the ensuing days, it is most convenient to give the 6·0 gm. of potassium chloride in a mixture with the 4·5 gm. sodium chloride, and these should be dissolved in not less than 2 litres of 2·5 per cent. dextrose, giving a potassium solution of a concentration of 40 mEq/l. (See Appendix A.) If this strength is exceeded, the solution will cause pain when running in and is liable to thrombose the recipient vein. Using a solution of this strength (2·25 gm. [40 mEq] NaCl and 3·0 gm. [40 mEq] KCl in 1 litre of 2·5 per cent. dextrose), the basic intake is then provided by administering each 12 hours two bottles (1·0 litre) of this electrolyte mixture and one bottle (0·5 litre) of 5 per cent. or 10 per cent. dextrose. (See later section for further details of intravenous potassium administration.)

This intake has now been given to a large series of patients undergoing many different types of operation, and has been found to give excellent results. A series of balance studies has been performed with this intake, and these confirm that it gives rise to no serious retention. Figure 18 shows the sodium balance and body-weight figures from a typical case undergoing partial gastrectomy for benign peptic ulcer and receiving this intake after operation. Following operation there was a steady fall in weight, showing that no water retention occurred, and the sodium balance also shows the absence of any retention of significant proportions. Figure 19 shows the potassium balances from 3 similar cases; as would be expected, all 3 cases were in markedly negative balance on the day of operation, but this loss was soon replaced, without there being any evidence that this level of potassium administration causes a dangerous retention.

While this basic intake is designed to minimise the effects of the various metabolic changes taking place after operation, it must not be forgotten that these changes none the less take place, and will be reflected in the blood and urine changes in all cases undergoing operation. The existence and significance of these changes must be constantly borne in mind, otherwise serious errors will be made. Their main practical applications can be summarised as follows:

(1) Diminished urine output, with a urine of high specific gravity and raised electrolyte (sodium and chloride) concentration is inevitable for 24–48 hours after operation and does not mean that the patient is short of water. Under no circumstances should an attempt be made to "force" a high urine output during this time, as this will only cause water retention and even water intoxication.

replacement of such losses is considered in detail in the following section. Many other difficulties may occur, but two common ones merit especial consideration, namely the problems presented by (*a*) an unexpectedly low urine output, and (*b*) the finding of lowered serum electrolyte concentrations. Either of these conditions may be due to some quite simple cause, or may be a manifestation of some serious derangement, and both can usually be elucidated quite simply.

Considering the problems presented by the patient who at some time 2-3 days after an operation is found to have a markedly reduced urine volume, it is, of course, essential in the first instance to make sure that there really is a low urine output, not simply a failure in bladder evacuation with retention. There are many possible reasons why a patient may develop an inadequate urine output at this time, but in practice it is usually due to one of four causes. First, it may be the result of an *inadequate intake*. This is most commonly seen in patients not on intravenous fluids, who for some reason have not drunk sufficient since operation. The patient will in general be making an otherwise uneventful recovery from operation and will show no untoward physical signs. The urine will have a raised specific gravity (above 1010), but will contain no abnormal constituents, and a simple review of the intake figures over the previous 24–48 hours will nearly always make the diagnosis clear. The fluid intake should be increased, if possible by mouth, and a concomitant rise in urine output will both confirm the diagnosis and settle the problem. (See Appendix B: Case 8.) Secondly, the low urine output may be due to *abnormal losses*, which have either not been replaced at all, or have been replaced inadequately. Such losses are usually overt and normally by vomiting or suction up a gastric tube. Depending upon the volume of the losses, the patient may or may not show signs of depletion (see later section), and will probably have some symptoms and signs from the condition causing the abnormal losses. Owing to the coincident changes due to the metabolic response to surgery, the urine findings will be variable and must be interpreted with caution (see p. 50). Again an analysis of the intake and output figures over the previous 24 hours or more will usually make the diagnosis clear, and the administration of the appropriate fluid (see p. 65) will replace the deficit and restore the urine output. In some cases of paralytic ileus difficulties may arise if the main loss is not overt but concealed, consisting of fluid accumulating in the dilated, atonic intestine. Indeed in some cases the first sign of a developing ileus is the sudden falling off in urine volume. In such cases a careful clinical examination will usually reveal the true diagnosis, and a cautious addition to the basic intake will be required.

Uncommonly the low urine output may be due, thirdly, to an

replacement of abnormal losses is decidedly more difficult in this period. For these reasons it is highly desirable not only that any deficiency should be corrected before operation, but also that the basic intake should be accurately maintained thereafter, so as to provide a standard baseline against which disturbances can be assessed and corrected.

By far the commonest difficulties in the post-operative period are those arising as the result of abnormal losses from the alimentary tract, as a result of paralytic ileus, fistula formation etc., and the

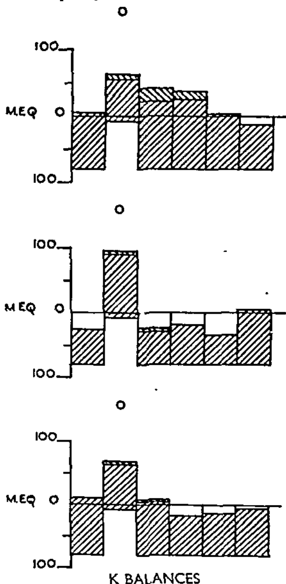


FIG. 19.—Potassium balances from three patients undergoing gastrectomy who received the standard intake recommended in the text.

unusually prolonged and *severe impairment of water excretion.* As explained previously, the post-operative impairment of water excretion usually only lasts some 24–36 hours, but in rare instances it may be prolonged for 48 hours or more. In such cases the patient may be making an otherwise normal recovery, or may show early signs of water intoxication. The urine will be scanty, with a raised specific gravity and electrolyte concentration, but with no abnormal constituents. The fluid balance charts will show the intake to have been adequate, with no abnormal losses. The condition is of importance as it carries with it a distinct risk of water intoxication, a complication discussed in full in a later section. If it is decided that the low urine output is due to this condition the intake must be restricted to not more than 1·0 l. per 24 hours until such time as a diuresis shows that the impairment of water excretion has worn off. If definite water intoxication has developed, more specific treatment may be required.

Again uncommonly, the low urine output may be due, fourthly, to *acute tubular necrosis,* by far the most serious cause of oliguria after operation. In severe cases there will be complete anuria, but if the renal damage is less there may be only a profound diminution in urine flow. But in all cases there will be a history of the incident precipitating the tubular necrosis, the commonest being either a severe fall in blood pressure during or after operation, or, fortunately rarely, the giving of a mismatched blood-transfusion. Further, in the great majority of cases there will be blood pigments, albumin and sometimes casts in such little urine as is formed. Early recognition of the condition is essential if treatment is to be successful, and the whole subject of the management of these cases is discussed in a later section.

From time to time difficulties may arise when, for one reason or another, the serum electrolytes are estimated after operation, and the sodium and chloride concentrations are found unexpectedly low —a problem which is discussed in full by Moore (1954) and Wynn and Rob (1954). In the great majority of cases this finding is simply a manifestation of the metabolic response to surgery, and is a perfectly normal finding under the circumstances. In such cases the patient will be making an uneventful recovery from operation, will have a normal urine output, and the balance charts will show a normal intake with no evidence of abnormal losses. This normal fall in the plasma sodium concentration may last for 7 days after operation and the concentration may go as low as 125 mEq/l.: it is characteristically accompanied by a rise in the serum potassium level to about 5·0 mEq/l. or slightly more. In that these changes are part of the normal response to surgery, they require no treatment and should be ignored.

Less commonly the finding of a low serum sodium concentration

after operation is indicative of either (*a*) an excess of water within the body, or (*b*) a deficit of sodium, leading to a relative excess of water, and these two conditions can usually be recognised and distinguished quite simply. If the low figure is due to an excess of water the patient will either be suffering from, or in danger of, water intoxication; the urine output will be low with a raised specific gravity, and the balance charts will show a normal or excessive intake, with no or insignificant abnormal losses. If a sodium deficit is the cause of the low concentration, there will usually be other clear clinical and biochemical evidence of salt depletion and there will be a history of abnormal losses, again usually from the alimentary tract. In either instance the treatment is clear and is discussed in detail elsewhere, but it cannot be stressed too strongly that, especially after operation, the finding of a low serum sodium concentration must not be taken *per se* as an indication of salt depletion and so for an increase in the salt intake.

Stress has previously been laid on the value of keeping accurate intake and output charts after operation, particularly if the patient is receiving intravenous fluids. It will be seen that with most of the problems arising at this time the correct diagnosis and management can be deduced by nothing more complex than considering the evidence provided by such charts, and in fact, taken in conjunction with the clinical state of the patient, they provide by far the surest guide to the management of fluid and electrolyte problems at this time.

DEHYDRATION

EFFECTS OF ABNORMAL LOSSES

In practice the maintenance of a patient in normal water and electrolyte balance is not difficult, provided the kidneys are functioning normally and there are no abnormal losses. However, if such losses occur, not only the maintenance of balance, but even the preservation of life may be difficult. In cases of intestinal obstruction, post-operative paralytic ileus, etc., the correction of depletion due to abnormal losses often constitutes the immediately urgent therapeutic problem, and in such cases the correct interpretation of the clinical findings and biochemical changes requires at least a working knowledge of the mechanisms of dehydration.

As seen clinically, such cases present a complex picture, but essentially this derives from two distinct causes, namely a water deficit and a salt (sodium) deficit, each of which results in fundamentally different changes. In addition there may well be other complicating factors, such as an acid-base disturbance or a potassium deficiency, but in the large majority of cases the important deficits are in water and sodium, and if these are replaced recovery will take place In clinical practice a pure water deficit or a pure salt (sodium) deficit is rarely seen, but a consideration of the differing effects of these pure losses, stressed by Marriott (1947, 1950), is an essential preliminary to an understanding of the complex clinical manifestations of dehydration.

Water deficiency, or primary dehydration, is occasionally seen in almost pure form in cases with markedly deficient intake, as in carcinoma of the œsophagus. The critical physiological effect of water deficiency is to cause a concentration, and rise of osmotic pressure in the extracellular space, resulting in a shift of water out of the intracellular space, and the restoration, to a varying degree, of the size and tonicity of the extracellular space (Fig. 20). Thus water deficiency causes essentially an intracellular depletion, and with this a loss of potassium from the body in an attempt to maintain intracellular isotonicity. Clinically the first and outstanding manifestation is thirst, which later becomes extreme, and is accompanied by a progressive dryness of the mouth and mucous membranes. In the later stages there is increasing weakness and mental change, leading finally to death in coma. In the early stages the blood changes are minimal, but later there is progressive hæmoconcentration, with rising hæmoglobin, hæmatocrit and plasma electrolyte

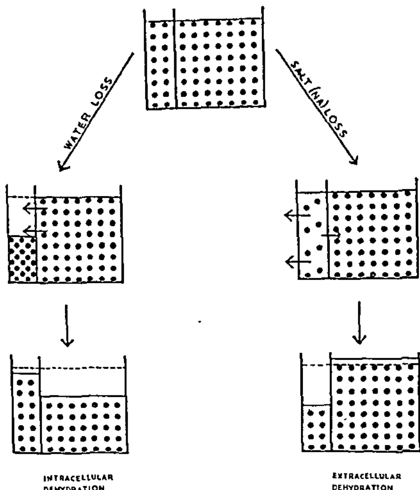


FIG. 20.—Diagrammatic representation of the differences between water and salt depletion The body water is represented as a tank, divided into the larger intracellular, and smaller extracellular spaces, the dots representing the concentration of electrolytes in the spaces, and the small arrows the shifts of water caused by alterations in osmotic pressure. Water loss causes intracellular dehydration, and salt loss extracellular dehydration, with a tendency to intracellular overhydration.

levels (Fig. 21). The kidneys have the impossible task of excreting, in minimal quantities of water, not only normal nitrogenous end-products, but also excess electrolytes, especially sodium and potassium; as a result the urine volume is low or even zero, with a high specific gravity and electrolyte concentration, and there will be nitrogenous retention, with a rising blood-urea figure.

Salt (sodium) deficiency, or secondary dehydration, is occasionally seen in pure form when mixed losses are replaced by water only. The critical physiological effect of salt deficiency is to cause a lowering of the extracellular osmotic pressure, leading to two dis-

tinct events. On the one hand there is a shift of water into the intracellular space, and on the other the kidneys excrete the relative excess of water in an attempt to restore extracellular tonicity. This latter effect is by far the most prominent, so that the final result of salt (sodium) deficiency is an extracellular depletion, with a

	WATER DEPLETION	SALT (NA) DEPLETION
CLINICAL	THIRST	DIMINISHED SKIN ELASTICITY ETC LETHARGY POSTURAL HYPOTENSION VOMITING PERIPHERAL CIRCULATORY FAILURE
BLOOD	RISING BL UREA RISING HB (LATE) RISING NA & CL (LATE)	RISING HB & PR RISING BL UREA FALLING NA & CL (LATE)
URINE	HIGH SPGR	LOW OR ABSENT CL

Fig. 21 —*Diagrammatic representation of the different effects of salt and water* depletion. In this diagram the tank represents only *the extracellular space*, divided into its two components, the larger extravascular or interstitial space, and the smaller intravascular or plasma space. The dots represent the electrolytes in solution, and the circles the red cells and/or plasma proteins. The arrows and containers on either side represent the urine output. Note (*a*) The low urine out-

in both water and salt depletion

tendency to intracellular overhydration (Fig. 20). In the extracellular space the brunt of the loss falls on the interstitial space, as the osmotic force of the plasma proteins maintains the intravascular volume at the expense of the interstitial. But in severe deficits the plasma protein osmotic force will no longer be adequate to maintain the intravascular volume, and consequently an increasing deficit in the circulating blood volume will develop.

Clinically, extracellular depletion differs markedly from the intracellular type. At an early stage there is loss of energy, progressing to apathy, and this is associated with obvious muscular weakness, sometimes with cramps. In sharp contradistinction to water deficiency, thirst does not occur, and there may well be nausea and vomiting (Marriott, 1947). On examination the outstanding physical signs are loss of skin elasticity, a "putty-like" feel to the muscles, lowering of eyeball tension and wrinkling of the tongue. As the plasma volume begins to diminish, there will be giddiness and faintness on standing, with a soft, compressible pulse of increased rate, and with postural hypotension. As the circulating volume becomes progressively smaller these cardiovascular signs become increasingly prominent, finally developing into the full picture of peripheral circulatory failure, with a cold, clammy, cyanosed skin, especially at the extremities, empty peripheral veins, a rapid pulse and a greatly lowered blood-pressure.

In the early stages of salt depletion examination of the blood will show little abnormality, but as the plasma volume decreases there will be increasing hæmoconcentration, with steadily rising hæmoglobin, hæmatocrit and plasma protein figures. Until late in the condition the plasma sodium and chloride concentrations will show little or no change, but later they will be lowered (Fig. 21). As the condition progresses kidney function is depressed, probably as a result of a fall in the glomerular filtration rate, with resulting nitrogenous retention and a rising blood urea. Provided the water intake is maintained the urine output remains adequate, with a low specific gravity, but its salt content (usually measured as chloride, see p. 71) is negligible or absent (Fig. 21).

Combined Water and Salt Deficiency

[illegible]

los[illegible]

one of combined water and salt loss—that is, a mixture of intra- and extracellular depletion—often with additional complicating factors. By far the commonest cause of dehydration as seen in surgical wards is an abnormal loss of fluids from the alimentary tract, either by vomiting, intestinal suction or fistula formation, as with an ileostomy. These alimentary fluids consist, not of water, but of

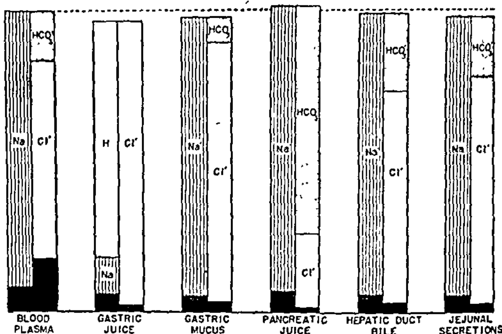


FIG. 22.—Stick graphs showing the composition of various alimentary secretions (from Gamble, 1950). Note that this figure is based on analyses of pure secretions, hence the electrolyte concentrations are higher than those in Table V, which is obtained by analysis of fluids as lost in clinical practice.

a solution of electrolytes in concentrations approximating to those in the plasma (Fig. 22; Table V). It will be seen that losses from the upper alimentary tract are more hypotonic than those lower down, largely owing to dilution by ingested water. In addition to these losses of electrolyte-containing fluid there is often an added pure water deficit, owing to continued insensible loss with a deficient water intake.

As a result of these two factors, in depletion due to abnormal losses from the alimentary tract the water loss is commonly in excess of the salt loss, and the clinical picture will be a combination of intracellular and extracellular dehydration in varying proportions. Usually the clinical signs of extracellular depletion predominate; lethargy, diminished skin elasticity and postural hypotension are almost invariably present, associated with sparse urine of high specific gravity and low or absent sodium and chloride content, hæmoconcentration, normal or slightly lowered plasma electrolyte concentration, and a raised blood urea. Thirst is often absent, even in the presence of extensive intracellular losses, possibly being masked by the mental depression resulting from the salt depletion. Even in the absence of a local cause in the alimentary system, nausea and vomiting are often present, and it has been suggested by Marriott

TABLE V

TABLE SHOWING THE ELECTROLYTE LOSSES IN FLUIDS FROM VARIOUS PARTS OF THE ALIMENTARY TRACT

Source	*Na mEq/l.*	*K mEq/l.*	*Cl mEq/l.*
Stomach	60 4 (9–116)	9·2 (0·5–32·5)	84 0 (7·8–154·5)
Small Bowel (M-A Suction)	111·3 (82–147 9)	4·6 (2·3–8 0)	104 2 (43–137)
Ileostomy (Recent)	129·4 (105 4–143·7)	11·2 (5·9–29 3)	116·2 (90–136 4)
Ileostomy (Adapted)	46	3 0	21·4
Cæcostomy	52·5	7·9	42·5
Formed Stool	<10	<10	<15
Bile	148·9 (131–164)	4 98 (2 6–12)	100 6 (89–117·6)
Pancreatic Juice	141·1 (113–153)	4 6 (2·6–7·4)	76·6 (54·1–95·2)

(From Lockwood and Randall, 1949)

The upper figure is an average, whilst those in brackets show the range of observed values.

(1947, 1950) that salt deficiency causes pylorospasm and gastric atony, thus leading to vomiting, a point further considered by Leger *et al.* (1955). In addition the cellular overhydration associated with salt deficiency is probably partly responsible for this symptom, which may lead to a vicious circle, with exacerbation of the salt deficiency. The importance of this in many surgical conditions needs no emphasis, and it must always be borne in mind that both vomiting and peripheral circulatory failure may be due to salt depletion.

Four further factors often exist, either singly or in combination, to complicate the picture—namely acid-base disturbances, alterations in kidney function, distributional shifts and potassium deficiency. In many cases these factors are not of significance in regard to therapy, but they demand consideration in that they may alter the clinical findings considerably.

Acid-base [illegible]
the [illegible]
re[illegible]
occur as the result of losses from the alimentary tract, and the magnitude of the water and salt loss which can occur from this cause has already been stressed. But the alimentary secretions do not contain acid and basic radicles in equivalent amounts, so that a differential loss of either one or the other may occur. If the sodium

loss predominates, there will be a resultant compensatory fall in the plasma bicarbonate, a fall in the CO_2 combining power (normal 24–32 mEq/l.), and acidosis. If, on the other hand, the chloride loss predominates, there will be a compensatory rise in plasma bicarbonate, a rise in the CO_2 combining power and an alkalosis.

It is to be noted that in each instance the alkalosis and acidosis are not caused by these alterations in the CO_2 combining power of the plasma, but that this alteration is a result of the compensatory respiratory and renal mechanisms by which the body maintains constant the pH of the blood. Of these two the respiratory factor is the more important, and its reserves are so great that it is only in extreme cases that a significant alkalæmia or acidæmia develops.

Clinical manifestations of acid-base disturbance are seen only in severe cases, the main signs being increased ventilation in acidosis, and in alkalosis latent tetany (tingling etc.) progressing later to frank tetany, sometimes with vomiting. It must be borne in mind that acid-base disturbances may also arise from causes other than excess loss of acid or base radicles, examples being respiratory failure with carbon dioxide retention (due to such causes as pulmonary emphysema or respiratory depression), renal failure and diabetic ketosis. *A common example in surgical practice is the ketosis of starvation* (especially in children) which may cause or exacerbate acidosis, though rarely to a severe degree unless there is also a very low urine output or impaired renal function.

The chemical structure of the various intestinal secretions is shown in Fig. 22. Loss of gastric juice, as in pyloric stenosis, will usually contain an excess of chloride, and severe alkalosis may easily develop (Fig. 23). However, in some cases of pyloric stenosis the associated gastritis may cause such a depression of acid secretion that the main loss consists of gastric mucus, and in these cases the loss of sodium and chloride will be essentially equivalent. This point was well illustrated by a case of carcinoma of the pylorus with stenosis studied in our wards, in which the gastric contents aspirated over a 48-hour period contained 109 mEq Na and 102 mEq Cl (see also Walker's (1949) figures). Biliary losses cause no acid-base *disturbance, but pancreatic juice contains a marked excess of sodium* (Fig. 1), and pancreatic fistulæ readily cause marked extracellular depletion with acidosis. In cases of intestinal obstruction or fistula the result will depend on the site of the obstruction or fistula. Thus an ileostomy or marked diarrhœa will cause an excess sodium loss and tendency to acidosis (Fig. 23), whereas a jejunal obstruction will cause no acid-base disturbance, as the mixture of gastric, biliary, pancreatic and intestinal secretions which is lost contains proportional amounts of sodium and chloride.

Kidney function is often markedly depressed in severe extracellular

depletion. As previously described, in the face of small to moderate deficits of sodium the kidney will excrete water, with the preservation of extracellular tonicity. However, with large deficits of sodium this mechanism breaks down, probably because the glomerular filtration rate, and with it the urea clearance, falls; as a result there is oliguria with water and nitrogen retention, a rapidly rising blood urea and a fall in plasma electrolyte concentration. It is as though there suddenly comes a time when the kidneys abandon the attempt to maintain tonicity, and concentrate instead on the preservation of volume. Associated with this failure of the kidneys to excrete

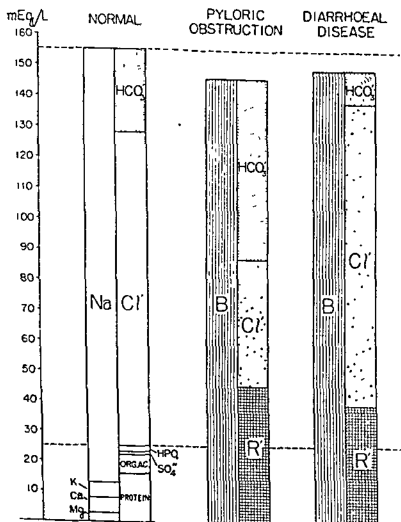


FIG. 23.—Stick graphs showing the effect of acid-base disturbances on the composition of the plasma. Pyloric stenosis gives rise to alkalosis, and diarrhœal disease to acidosis. "B" is largely composed of sodium, and "R" represents organic acids, etc. (from Gamble, 1950)

water and nitrogen there is at the same time a failure to excrete any ions in excess as the result of a differential loss, so that acid-base disturbances become accentuated. As stressed by Moyer (1947) the main importance of this depression of renal function is in relation to treatment, since the administration of glucose solutions instead of saline can only do harm and intensify the extracellular hypotonicity.

Distributional shifts, or concealed losses, within the extracellular space are common, though infrequently sufficiently large to be clinically significant. There is constantly a shift of extracellular fluid and protein into any damaged tissue, usually without overt loss, though in cases of burns this loss becomes manifest and of great significance. Two other examples of concealed losses which occur commonly and may well be of significant size are the shift of fluid into the actual bowel wall above the site of an obstruction, and the accumulation of fluid in the peritoneal cavity in cases of peritonitis. In a recently treated case of perforated gastric ulcer just over 700 ml. of fluid was aspirated from the peritoneal cavity, containing per litre 136 mEq Na, 110 mEq Cl and 6·5 mEq K. This figure does not, of course, represent the total quantity of peritoneal exudate, but gives some idea of the amount of fluid which may be temporarily *lost from the body from a functional point of view.*

The other important cause of distributional shifts in surgical patients is protein deficiency, as hypoproteinæmia will cause an excessive accumulation of fluid in the interstitial space. In severe cases dependent œdema, even ascites, will develop, but in less severe cases the results, though not obvious, may be just as serious, owing to the formation of œdema in wounds, at intestinal suture lines, etc. The administration of excessive amounts of salt is, of course, additionally dangerous in such cases of hypoproteinæmia, as it readily precipitates œdema formation.

Potassium deficiency is a common accompaniment of severe dehydration, and may cause significant clinical complications, which demand active treatment. The subject is dealt with in detail in a later section.

REPLACEMENT OF ABNORMAL LOSSES

The management and replacement of abnormal losses is one of the most difficult problems in fluid balance: there is no one single test to show the size of a deficit or the adequacy of replacement, and the only sure guide to a surgeon faced with these problems is constant, careful observation of the patient and his response to treatment In practice, the problems presented by abnormal losses arise in two different sets of circumstances. The losses may occur whilst the patient is actually under observation (observed losses), or the losses may have occurred before the patient is seen, and only their

effects can be observed (pre-existing losses). These two situations demand entirely different treatment, and will be considered separately.

Replacement of Observed Losses

Observed losses present by far the easier of the two problems: common conditions in which they arise are the post-operative development of paralytic ileus with loss by vomiting or gastric suction, the development of fistulæ of various types and the loss occurring during the passage of a Miller-Abbot tube for intestinal obstruction. It is to be noted that, almost invariably, observed losses consist of fluid containing a virtually isotonic concentration of electrolytes, and it is only rarely, as with excessive sweating, that losses occur which are grossly hypotonic.

The cardinal rule in the treatment of observed losses is the volume-for-volume replacement of these losses by normal saline (except in the rare cases mentioned above), a doctrine originating from the pioneer work of Coller and Maddock. For its success this form of treatment depends on two main supports, namely that the losses should be carefully measured, and that the replacement fluid is given over and above the basic requirements. Commonsense is required in deciding when abnormal losses are sufficiently large to require specific replacement. Provided the patient's basic intake approximates to that described previously it is usually unnecessary to add to it unless the losses exceed 250 ml. per 12 hours, or continue at this rate for many days. Without giving rise to constant trouble it is not possible to replace the losses immediately they occur; it is simpler and quite satisfactory to work 12 hours in arrears—that is to say, twice a day the losses during the previous 12 hours are added up, and in prescribing the fluid intake for the next 12 hours an appropriate volume of normal saline is added to the basic intake. If fluids can be taken by mouth, normal saline flavoured with fruit juice can be given, or more simply the ordinary drinks increased by [illegible] o give [illegible]. fluids [illegible] ust be administered, the volume being correct to the nearest ½ litre (500 ml.). Whilst volume-for-volume replacement is being carried out, it is essential to measure not only the losses but also the urine output, as this will provide a good guide to the adequacy of the replacement. Distributional shifts, as for instance into the bowel wall and lumen in intestinal obstruction, are often quite large, and if the replacement is insufficient this will be shown by a falling off in urine volume, indicating the need for increasing the intake. Estimations of the urine chloride concentration and specific gravity are also

important checks, but if the replacement is being carried out in the immediate post-operative period, then the influence of the post-operative metabolic changes must be carefully borne in mind when considering the significance of these findings. (See Appendix B, Case 2.) In cases in which the observed losses are very large, repeated estimations of the hæmoglobin concentration and, especially in children, repeated weighing form valuable additional guides.

Objections can be, and are, made to this simple scheme of treatment, on the grounds that the contents of the alimentary tract—the commonest source of abnormal losses—consist not of normal saline but of a fluid which is usually hypotonic, contains sodium and chloride in varying proportions, and also contains potassium. Before considering these objections in detail, it is important to stress the value of simplicity in drawing up schemes for handling these problems; it may well be that a simple schedule of treatment, though not biochemically ideal, is yet the treatment of choice if it demands only a limited number of different fluids, a minimum of laboratory control, and is in practice successful.

Considering first the point that the alimentary losses do not contain equivalent amounts of sodium and chloride, so that their loss may lead to acid-base disturbances (see below), there is no doubt that this danger has been over-exaggerated. If these losses are replaced by normal saline the kidneys will excrete the ion in excess, provided the urine volume is adequate, thus emphasising again the need to maintain, during volume-for-volume replacement, the basic intake, which is designed to give a urine output of 1500 ml. per day. A good example of this renal adjustment was provided by the case of pancreatic fistula previously referred to. This boy was losing up to 1 litre of pancreatic fluid daily, containing Na 155 mEq/l. and *Cl 57 mEq/l.: he was treated by additional fluids by mouth, together* with 10 gm. of NaCl daily in gelatin-coated capsules, and his urine was found to contain Na 66 mEq/l. and Cl 181 mEq/l. It would, no doubt, have been more biochemically exact to have administered a suitable mixture of sodium chloride and sodium lactate, but these figures show that such a refinement in treatment is not necessary, provided the kidneys are supplied with adequate water.

There is, however, more substance in the other two objections that the fluid lost is hypotonic and contains potassium. There is no doubt that when losses are large potassium must be given (see special section). As regards the hypotonicity, it is not essential to modify treatment to meet this point. In the case reported in detail in Appendix B (Case 5) the intestinal loss amounted to just over 4 litres per day, and this was replaced volume-for-volume with normal saline, the only adverse clinical effect being the production of slight sacral œdema by the fifth day (which was in part due to the excessively

large basic salt intake given). Losses greater than this are unlikely to be met with, and a form of treatment which will control such a situation adequately should not be modified without good reason. Nevertheless, it is probably wise to modify the method of replacement when losses are large. Several complicated formulæ have been proposed for making this modification. One of the simplest and best is shown in Table VI, taken from Lockwood and Randall's paper

TABLE VI

SUGGESTED METHOD FOR VOLUME-FOR-VOLUME REPLACEMENT OF LOSSES FROM THE ALIMENTARY TRACT

Source of Loss	Type of Fluid		
	A	*B*	*C*
Stomach	33 per cent.	67 per cent.	—
Small Intestine (Intubation)	20 per cent	70 per cent.	10 per cent.
Ileostomy	10 per cent.	75 per cent.	15 per cent.
Biliary Tract	—	67 per cent.	33 per cent.
Pancreatic Juice	—	50 per cent.	50 per cent.

(From Lockwood and Randall, 1949)

A = dextrose in water.
B = dextrose in normal saline.
C = M/6 sodium lactate.

None of these fluids contains potassium, which must be added when necessary (see text).

(1949), whilst Cooke and Crowley (1952), approaching the problem from a different angle, have described the successful use of two com-
[illegible] t of gastric
[illegible] modifica-
[illegible] of the two
following rules give excellent results:

(1) If in any given 24-hour period the observed losses exceed 2·0 litres, the fifth ½ litre should be replaced with 5 per cent. dextrose: and so on in multiples of five.

(2) If in any given 24-hour period the observed losses exceed 2·0 litres, or if the losses continue for more than 48 hours, the replacement fluid should contain potassium, unless the basic intake contains this ion in adequate amounts. The potassium should only be given subject to the precautions set out below (see special section).

To conclude, when observed losses occur, they should be replaced,

over and above the basic intake, volume for volume by normal saline, save when they exceed 2 litres (4 pints) per 24 hours, when the type, not quantity, of fluid should be modified as suggested above.

Replacement of Pre-existing Losses

Pre-existing losses present a more difficult therapeutic problem, as exact information about the volume of the losses is rarely, if ever, available. Further, at the present time there is no single test or estimation which will give an accurate figure for the size of the deficit. Many tests and formulæ, of varying degrees of complexity, have been suggested for this purpose, but there are so many variable factors in any given case that so far none has been found to stand up to the test of practical experience. One of the best known of these formulæ was the "clinical rule" proposed by Coller and Maddock (1940), by which a calculation was made of the extracellular deficit based on the plasma chloride level, but after experience in its use these same authors (Coller *et al.*, 1944) realised its inadequacy and rescinded their previous advice. Recently Black (1953) has suggested a simple formula for calculating the sodium deficit, based on the serum sodium concentration. Whilst admitting it gives only "a crude measure of the saline requirement," Black claims this formula is of some value, but more extensive experience is required to assess its full worth.

Estimation of the size of the extracellular space by use of the thiocyanate method (Crandall and Anderson, 1934) is an investigation that has been practised and recommended by some workers (Walker, 1949). Quite apart from the questionable accuracy and relative complexity of this observation, it is doubtful whether the information it provides is reliable. Trials of this technique in our unit failed to give consistent results in normal subjects, and gave quite misleading results in depleted patients, probably owing to alterations in membrane permeability during dehydration. Other substances, such as urea, radioactive sodium, etc., have been used to measure the size of the various fluid compartments, but so far none has proved both simple and reliable enough for routine use.

In practice the size of the deficit of both water and electrolytes must be assessed on the basis of a careful clinical and biochemical examination of the patient, aimed at detecting the evidences of water and salt depletion previously described. The importance of the clinical part of this process must not be underestimated. In cases of slowly developing or long-standing depletion the history is of little value, but in acute cases it is often possible to reach an approximate figure for the amount lost in, say, vomiting, and also to get an accurate idea of the length of time during which insensible loss has

been continuing with inadequate or absent replacement due to interference with intake. On examination, the main features to be observed are the pulse volume, the blood pressure, the state of the skin and tongue, eyeball tension, muscular tonicity and the degree of apathy, and the more important of these are discussed below. Valuable biochemical information can be obtained from the blood and urine. Of the various blood and plasma estimations available, by far the most instructive are the hæmoglobin and/or hæmatocrit, the plasma proteins and the blood urea; plasma electrolyte concentrations and the alkali reserve are often helpful but rarely essential. From the urine, the most helpful information is provided by its volume, specific gravity and electrolyte concentration. The evidence presented by these three facets of a case—namely clinical evidence, blood and plasma estimations, and urine estimations—needs critical examination if the deductions are to be reliable, and the considerations to be borne in mind are discussed below under these main headings. As a preliminary to this it is important to stress that, apart from their contribution to the primary assessment, these initial observations are of incalculable value in giving a base-line against which the effects of treatment can be observed, and for this reason alone a minimum of estimations should be made in all cases.

Clinical Observations. Absence of thirst is no evidence that the patient is not depleted, and even in cases with marked intracellular deficiency, thirst may be lacking if there is also an extracellular deficit. Apathy and muscular weakness indicate appreciable salt deficiency, and muscular hypotonicity is characteristic of hypokalæmia. With marked extracellular deficiency the blood volume begins to diminish, leading in time to vasoconstriction, falling blood pressure, and eventually peripheral circulatory failure. Initially the only sign of this process is postural hypotensi [illegible] sure s [illegible] Later, [illegible] tension, with a rapid pulse, clammy extremities, and peripheral cyanosis; this condition is indicative of severe extracellular depletion (a deficit of 5 litres or more), and it is essential to recognise its association with salt depletion, otherwise serious therapeutic errors may be made.

The state of the skin is best appreciated by examining that over the antecubital fossa or lower neck; in elderly patients or those who are malnourished, skin elasticity is often diminished even when they are normally hydrated, so that in these circumstances diminished elasticity must be interpreted with care. Similarly, in assessing the significance of a dry tongue and mouth, care must be taken to see that the patient has not just been sleeping or dozing with his mouth open.

Blood and Plasma Estimations. The hæmoglobin, hæmatocrit and plasma protein levels all give essentially the same information, in that they vary with the degree of hæmoconcentration. In general they are the most useful of all the blood tests, particularly when repeated at intervals during the course of treatment (see Appendix B, Case 3). In assessing the significance of these figures, care must be taken to bear in mind the effect of any pre-existent anæmia or hypoproteinæmia, especially when planning treatment (*vide infra*). Any marked degree of water or salt shortage leads to nitrogen retention, so that the blood urea is a most useful and sensitive index of the degree of depletion and the efficacy of replacement: together with the hæmoglobin it is one of the estimations which should never be omitted. Estimations of the plasma sodium and chloride concentrations are essential in complex cases, but in most cases are not obligatory and may even be confusing. Severe degrees of depletion can readily exist with normal plasma electrolyte levels (see Fig. 20), so that a normal plasma chloride figure must not be taken as indicative of normal hydration: it is only in the later stages of depletion, when the diagnosis should be obvious without their aid, that the plasma electrolyte levels show significant falls. This point, emphasised by Sanchez-Vegas and Collins (1946) in their study of pyloric stenosis, was nicely illustrated by a similar case of ours. On admission the plasma concentrations of Na and Cl were 140 mEq/l. and 103 mEq/l respectively, yet during the next 72 hours, during which time rehydration was carried out, this man retained just over 3½ litres of water, 600 mEq Na and 470 mEq Cl, without any significant alteration in his plasma electrolyte concentration.

In view of the fact that acid-base disturbances can usually be ignored, the alkali reserve (CO_2 combining power) is an estimation of limited value, and need only be done in complicated cases. In such cases the plasma sodium and chloride concentrations will probably be estimated and usually give the same information in different form, but a double check may be valuable.

Urinary Observations. Some idea of the urine volume over the preceding few hours can nearly always be obtained, together with a measurement of the volume passed during the first 2–3 hours of the patient's stay in hospital. The specific gravity of this first specimen should always be recorded, care being taken to exclude glycosuria as the cause of any significant rise. A very low urine output, with a specific gravity above 1015, is indicative of water depletion, and these two observations are amongst the most significant provided by the urine. An estimation of the urine chloride concentration is also of great value, but in assessing its significance three points must be borne in mind. First, the extracellular ion of cardinal importance is sodium, so that the urinary sodium concentration is the desired

figure. In the absence of a flame photometer this information is obtained only with difficulty, whereas the urine chloride is quickly estimated. Accordingly the urine chloride concentration is frequently performed, and then interpreted as giving information about the sodium concentration: in most cases this assumption is valid, but care must be taken that a coincident acid-base disturbance is not disturbing the normal urinary sodium-chloride ratio. Secondly, the figure of real significance is the absolute amount of sodium or chloride passed in the urine, rather than the concentration, which varies with the volume. Accordingly oliguria with a very low concentration of chloride is significant of depletion, but the same concentration in the presence of polyuria would be consistent with normal balance in respect of chloride, so that the volume of urine passed must always be borne in mind when assessing the significance of chloride concentration. Thirdly, if the Fantus test, advocated and popularised by Marriott (1947), is used in estimating the urine chloride concentration, great care must be taken to see that the glassware used is scrupulously clean, otherwise grave inaccuracies will occur. Provided these points are borne in mind, the urine chloride estimation is a most valuable test. Normally the urine chloride concentration varies considerably but does not fall below 50 mEq/l. (175 mgm. per cent.) save in the presence of a marked diuresis, so that a concentration below this figure is indicative of extracellular depletion, and with any severe deficiency there will be a complete absence of salt in the urine.

Summarising, the assessment of the size and type of the deficit depends, then, on a clinical and biochemical survey of the patient, the object being to dissect out from all the available evidence the critical stigmata of water and salt depletion. Care must be taken not to become overwhelmed in an excess of data, much of which often appears to be conflicting. The essential features of this survey are a careful clinical examination, an estimation of the hæmoglobin and blood urea concentrations, and a knowledge of the urine output, its specific gravity and electrolyte (usually chloride) content. In the great majority of cases this will suffice to detect any significant depletion, and it is only in complex cases that the estimation of serum electrolyte concentrations etc. is essential. In reaching the final estimate, which in any event can only be provisional, two useful figures should be borne in mind, namely that when dehydration is obvious the deficit amounts to one-fifteenth to one-twelfth (6 per cent.) of the body weight (Darrow, 1940), and further that when the dehydration is chronic rather than acute, approximately one-half of the deficit is intracellular—that is to say, an average-sized man (70 Kg.; 11 stone) presenting obvious dehydration needs at least 4 litres of fluid for replenishment, of which at least 2 litres must

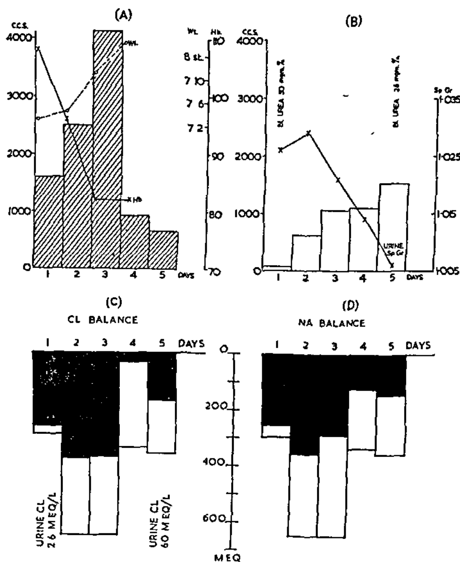


Fig. 24.

consist of normal saline. In many cases deficits considerably larger than this are seen, and in addition it must not be forgotten that there will probably be a coincident potassium deficiency, the diagnosis and management of which is discussed in the next section.

Once an assessment of the size of the deficit has been made, replacement can begin, the route used depending on the circumstances of the case. Whilst this replacement is going on the basic daily allowance must also be given; as this amounts to 3 litres per 24 hours, excessively rapid administration is called for if an attempt is made to make replenishment complete in 24 hours, so that if the

FIG. 24 (opposite).—Data obtained in the replenishment of a case of pyloric stenosis: throughout the period illustrated the effective intake was entirely intravenous, as the gastric aspirate equalled or exceeded the amount drunk. Day 1 was not a complete 24-hour period, so the figures on this day are low.

A—represents the water retained, calculated by subtracting the urine output, gastric aspirate and insensible loss (taken as 1500 ml. per 24 hours) from the total intake. The weight gain and hæmodilution resulting from this water retention are clearly shown.

B—shows the increase in urine volume (blocks) and fall in urine specific gravity as rehydration proceeded. Note the fall in blood urea resulting from re-establishment of the urine output.

C—shows the chloride balance: the output is not broken down into gastric and urinary moieties, and the resulting balance is blocked in (see Fig. 5). The greater part of the chloride output was actually gastric loss. The initial and final urine chloride concentration is also shown.

D—shows the sodium balance.

Note the large size of the deficit, as shown by the water and electrolyte retention

deficiency is at all marked the replacement should be carried out over 48–72 hours. As the initial assessment is necessarily only approximate, careful observations must be made during the course of replenishment, and every case should be considered as a therapeutic experiment, the response to treatment giving in many ways more information about the extent of the loss than the original assessment. Herein lies the value of the initial observations of hæmoglobin, etc., which gain immeasurably in value when compared with similar observations repeated after the administration of known amounts of water and electrolytes. There is no short-cut in the treatment of pre-existing losses, and the whole key to success lies in the initial assessment followed by regular observations of the effects of the treatment and modification of further administration as required (see Appendix B, Case 3).

When a depleted patient is given water and salt, the body uses these in a definite order of priority, which enables the surgeon to judge accurately when replenishment is complete. Insensible loss makes the first call on the fluid administered; the remaining fluid is used for replenishment of the body spaces, and it is only when this is complete that there will be available to the kidneys water and salt to provide a urine of normal volume, specific gravity and electrolyte composition. Accordingly, treatment may be said to be complete when there are no longer any clinical signs of depletion, when the hæmoglobin and plasma electrolyte concentrations are stable within normal limits, when the blood urea has fallen to a normal level and when the urine volume exceeds 1000 ml. per day with a specific

gravity of less than 1010 and a chloride concentration in excess of 40 mEq/l. (Fig. 24). All these points should be noted at intervals not exceeding 24 hours, and then treatment can be suitably modified as replenishment progresses. In practice it will usually be found that the last of these criteria to return to normality is the urine chloride concentration; commonly, all clinical signs of depletion disappear and a good urine output is established whilst the urine chlorides remain negligible. For this reason the main use of the urine chlorides, after the initial assessment, is to act as a "fine adjustment" in respect to saline administration (Appendix B, Case 3).

During the replacement of pre-existing losses, not only must the basic intake be maintained, but if the abnormal losses continue they must also be replaced on a volume-for-volume basis (Appendix B, Case 3). Bearing in mind that the basic intake is 3 litres per day, that a deficit of 3–4 litres may well exist, and that observed losses can easily amount to over 1 litre per 24 hours, it is apparent that on occasions large volumes of fluid will be required. Such considerations also stress the importance of replacing abnormal losses as soon as possible, so as to prevent the development of severe depletion and avoid the necessity for giving very large quantities of fluid in short periods of time.

Just as in the volume-for-volume replacement of observed losses, so in the treatment of pre-existing losses, acid-base disturbances can usually be ignored as they will correct themselves provided the urine flow is re-established. The only exception to this arises in the treatment of severe acidosis. The concentration of chloride in normal saline (160 mEq/l.) is considerably higher than that in plasma (95–100 mEq/l.); in acidosis the chloride ions are already in relative excess and the administration of saline, whilst replacing the sodium deficit, may lead to such an excess of chloride ions that the kidneys, especially as their function is likely to be depressed, are unable to excrete them fast enough to prevent an exacerbation of the acidosis. Under these circumstances at least part of the loss should be replaced by one-sixth molar sodium lactate (165 mEq/l.). In cases of alkalosis a special fluid is rarely required, as the concentration of sodium in saline and plasma is virtually the same. We have treated a case of pyloric stenosis, with alkalosis sufficiently severe to cause tetany, quite satisfactorily with saline alone. If it is desired to use a special fluid, 0·85 per cent. ammonium chloride (165 mEq/l.) is a safe and convenient solution.

During the replacement of pre-existing losses, care must be taken that a dangerous degree of anæmia and hypoproteinæmia is not unmasked. The hæmoconcentration resulting from extracellular deficiency may give rise to a misleadingly high hæmoglobin figure which falls rapidly as treatment progresses, and a dangerous anæmia

may be precipitated. This point is well illustrated by Case 3, Appendix B. This danger should accordingly be watched for, and where there is evidence of anæmia, blood transfusion should be given in appropriate amounts. The potential dangers of precipitating a marked anæmia were seen in a case recently observed of a 78-year-old clergyman with pyloric stenosis due to carcinoma, causing obvious severe dehydration. On admission, the blood urea was 144 mgm. per cent. and the hæmoglobin 43 per cent. During the next 48 hours the deficit was largely replaced by intravenous fluids and during this time the patient gained 10 lb. in weight, showing that he retained at least 4½ litres of water; he also received during this time 3 pints of blood, yet at the conclusion of this time his hæmoglobin figure had fallen slightly. It is obvious that if no blood had been given the process of replenishment would have led to an acute, severe anæmia. A similar danger exists in relation to potassium deficiency (Appendix B, Case 4), and demands equal attention; the whole problem of the administration of potassium to cases with pre-existing losses is discussed in a later section.

In cases with very severe depletion, with definite lowering of the plasma electrolyte concentrations, great difficulty may be experienced in raising them again to normal levels, even when all other signs of deficiency have disappeared. Various reasons, such as an associated potassium deficiency, have been suggested to explain this difficulty, which remains to be clarified. It may well be that the difficulty lies in the fact that in such cases the electrolyte deficiency is so great in proportion to the water lack that replacement fluids of a concentration greater than normal are required. The idea of treating hypotonic states with hypertonic fluids is certainly attractive, and Sanchez-Vegas and Collins (1946) used 3 per cent. saline with great success in the treatment of dehydration due to pyloric stenosis. In his Goulstonian Lectures, Black (1953) discusses this point and reports that he has given 5 per cent. saline intravenously to 4 sodium-depleted patients with marked clinical improvement and no untoward effects, and we have on several occasions used 2 per cent. saline with great benefit. But in general the use of hypertonic solutions has not received adequate consideration, and the subject would appear to demand more active attention than it has received in the past.

One further danger remains to be mentioned. Great care must be taken not to treat severe salt deficiency with hypotonicity by the [illegible]

[illegible] intoxication. Moyer (1947) emphasised this danger and ascribes the dangerous results to depressed renal function. This is probably only a contributory factor, in that it causes retention of

the administered water with further dilution of the extracellular ions. This increasing hypotonicity will cause a shift of water into the cells (Fig. 20), and it is this movement which leads to cellular overhydration and possibly water intoxication. Whatever the exact mechanism the danger is undoubtedly present, and the paradox of a seriously depleted patient suffering from water intoxication is not unknown. Accordingly, in the presence of severe extracellular depletion, normal saline must be administered to restore, at least in part, the extracellular volume before attempts are made to obtain a good urine flow with glucose solutions.

Finally, *the importance, whenever possible, of completing replenishment before operation cannot be overstressed*. Apart from the obvious disadvantages of operating on a water-deficient, salt-depleted patient, the difficulties of replenishment are much increased after operation. The control of replacement largely depends upon biochemical observations, especially the urine specific gravity and electrolyte concentration, which always undergo specific changes after operation, so that if replacement is attempted during this period it is difficult, if not impossible, to decide whether, say, a lowered urine chloride concentration is due to the operation itself or to inadequate replenishment (see Appendix B, Case 2). Under these circumstances only clinical guides are left, and every effort should be made to avoid this difficulty, though unfortunately urgent operations are sometimes required before replacement can be properly carried out.

POTASSIUM DEFICIENCY

ONLY within recent years have disturbances of potassium metabolism been recognised as being of importance. Previously it had been thought that clinically significant losses or increments of potassium rarely, if ever, occurred, but it is now known that important changes in potassium metabolism follow all major operations, and that potassium deficits sufficient to cause serious clinical states occur not infrequently. Study of this important new aspect of electrolyte balance was stimulated originally by Darrow's work on infantile diarrhœa, and the introduction of the flame photometer has undoubtedly facilitated research in this field during the last few years.

The total amount of potassium in a normal 70 Kg. (11 stone) man is 3400 mEq, of which only 2 per cent. (68 mEq) are in the extracellular space (Moore and Ball, 1952). Potassium forms the main intracellular cation, and is present in the intracellular fluid at a concentration of approximately 150 mEq/l. In contrast to this, the concentration of potassium in the extracellular fluid is only 4·0–4·5 mEq/l. Accordingly, the intracellular potassium forms a large reservoir on which the extracellular space can draw when losses occur. The average dietary intake of potassium is 75–100 mEq (3–4 gm.) per 24 hours, of which only some 10 per cent. is lost in the stools, whilst the daily urine loss is 50–75 mEq (Lans *et al.*, 1952). Sweat contains only minimal quantities of potassium. Whilst potassium is the predominant intracellular ion, it does not appear to be as indispensable as is sodium within the extracellular space. Within ill-defined limits sodium can replace potassium inside the cells (Newburgh, 1951), so that potassium deficiency will cause sodium retention, the so-called "Bunge phenomenon" (Gamble, 1951). But this interchangeability is not limitless, and animal experiments suggest that it leads to definite derangements of cell metabolism (Newburgh, 1951).

POST-OPERATIVE DISTURBANCES OF POTASSIUM METABOLISM

After operation there is an increased urinary loss of potassium, maximal on the day of operation and returning to normal within the next 48 hours. This point was investigated by Berry, Iob and Campbell (1948), who made observations on a group of patients undergoing either herniorrhaphy or abdominoperineal resection of the rectum, and receiving no potassium during and after operation.

Their results showed that the potassium loss during the 30 hours covering and immediately after operation ranged from 30–119 mEq for the whole group, and was distinctly greater after resection of the rectum than after herniorrhaphy. They also showed that this urinary potassium loss was greater than could be accounted for by the post-operative nitrogen excretion, and that it was increased by infusions of saline rather than dextrose. Investigating the same point, Randall *et al.* (1949) confirmed these results, and their figures showed that the post-operative urinary potassium loss is roughly proportional to the severity of the operation. In a further group of patients these same workers showed that the intravenous administration of some 50 mEq potassium each 24 hours does not prevent a potassium diuresis and negative balance on the day of operation and first post-operative day, but that thereafter there is a slight retention of potassium. Indeed, it would appear that it is not possible to prevent a negative potassium balance during the first 48 hours post-operatively, even by giving considerable amounts intravenously. Figure 19 shows the potassium balance in 3 cases and illustrates clearly a negative balance for 48 hours despite a high level of potassium intake. No information appears to be available concerning the results of giving potassium in greater amounts than this over the period of operation.

In recent years this potassium diuresis has received much attention, and was carefully studied by Moore and Ball (1952), who lay stress on the relative excess of potassium to nitrogen in the urine during the period in question. This relationship is usually expressed in terms of the K:N ratio, which is normally 2·7–3·0. When tissue is broken down, the liberated potassium and nitrogen are excreted in the urine in proportional amounts, giving a K:N ratio within normal limits. A rise in the K:N ratio, such as is found during the potassium diuresis after operation, shows that intracellular potassium is being liberated at a greater rate than tissue is being broken down. Accordingly the post-operative potassium diuresis must be indicative of a mobilisation of intracellular potassium, which then overflows into the urine, and the findings of Winfield *et al.* (1951) are interesting in this connection. They examined a series of 12 patients undergoing extensive operations, the average urinary potassium excretion during the first 48 hours after operation being 163 mEq. From these same patients they analysed a series of muscle biopsies, and showed that during the period of operation there was a fall in muscle potassium of approximately 50 per cent. in muscles near the operation site, but of only approximately 10 per cent. in distant muscles, suggesting that the potassium diuresis and raised K:N ratio may well be due to mobilisation of intracellular potassium from tissues adjacent to the field of operation.

This post-operative potassium diuresis is almost certainly adrenocortical in origin, and its occurrence on the day of operation is one of the strongest pieces of evidence indicating adrenocortical release on this day (see previous section). In the great majority of patients who return to a normal diet shortly after operation, this potassium diuresis is of no clinical importance, but in the minority who require intravenous fluids for several days post-operatively it may be of crucial significance in relation to the development of definite potassium deficiency.

POTASSIUM DEPLETION

Potassium deficiency states are of far greater practical importance to the surgeon than the loss on the day of operation, and modern work shows that they occur quite frequently. Indeed, it is probable that at the moment undue attention is being paid to potassium derangements, with deflection of interest from sodium and water balance, which remain the considerations of outstanding importance in most cases of electrolyte imbalance. Nevertheless, cases undoubtedly are seen in which critical deficiencies of potassium occur, and in which recovery may not take place unless this deficiency is replaced.

In a valuable review of the subject Moore and Ball (1952) describe three different types of potassium depletion. In "balanced starvation" there is a loss not only of potassium but of all the other constituents of the cell matrix in proportion, thus giving a normal urinary K:N ratio (3·0), and no alteration in the plasma potassium level. In "differential intracellular depletion," on the other hand, potassium leaves the body faster than the other constituents of the cell matrix, leading to a disproportionate potassium loss, with a raised urinary K:N ratio. This is the type of loss characteristically seen after an operation, or following intravenous glucose and saline infusions; it is usually not accompanied by a significant fall in the plasma potassium concentration but, by depleting the intracellular stores, it renders the body vulnerable to further losses and the development of hypokalæmia. "Extracellular depletion" is the condition arising when there is a loss of potassium from the body at a rate or to an extent beyond which it is capable of being replaced from the intracellular space, and clearly leads to a falling plasma level and hypokalæmia. This condition is usually caused by losses in the urine and from the gastro-intestinal tract, and its development is facilitated by pre-existing "differential intracellular depletion." Save on strictly theoretical grounds, the differences between the last two of these types of depletion are slight, and it might well be easier to consider "extracellular depletion" as simply the advanced stage of

"differential intracellular depletion," but the distinction between these latter two and "balanced starvation" is important and should be clearly borne in mind when considering cases in negative potassium balance.

The basic effect of potassium loss from the body is to cause intracellular hypotonicity. However, as mentioned previously, potassium is replaceable within limits by sodium, so that potassium

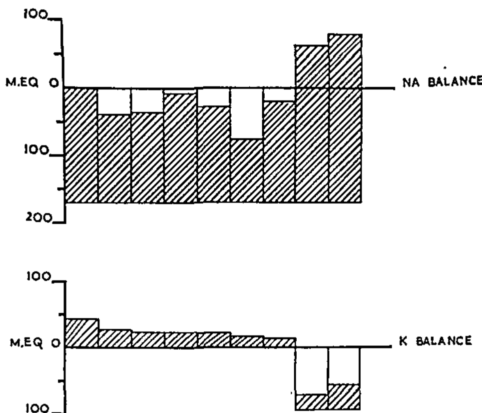


FIG. 25.—Sodium and potassium balances from a patient receiving a potassium-free drip-feed for 7 consecutive days. Note the sodium retention during this period, followed by an excretion of sodium when potassium is given

deficiency leads to sodium retention. This was clearly shown by Black and Milne (1952) and by Fourman and Ainley-Walker (1952), both of whom produced potassium deficiency in normal subjects by the use of ion-exchange resins. This point was investigated (Le Quesne and Lewis, unpublished work) in 3 patients with peptic ulceration who were given a potassium-free drip-feed for several days; all 3 showed sodium retention, the results in one case being shown in Fig 25, though the degree of sodium retention bore no constant relationship to that of potassium depletion. The influence of

this phenomenon on post-operative salt retention has already been discussed. Potassium deficiency is also accompanied by marked depression of muscular tonus and activity, affecting all types of muscle, and it is this which gives rise to the outstanding clinical signs of the condition, such as hypotonicity of the limbs, abdominal distension, and changes in the electrocardiogram (see below). Probably the essential cause is an alteration in the intracellular-extracellular ratio of potassium, thus accounting for those cases of potassium deficiency occurring with a normal plasma potassium level.

Ætiology of Potassium Deficiency

The outstanding feature in the production of potassium deficiency is a zero or negligible potassium intake over a period of days, particularly when this is accompanied by increased potassium loss either in the urine and/or from the gastro-intestinal tract (see Appendix B, Case 6). Accordingly, hypokalæmia is most commonly seen in the post-operative period and in abdominal surgical cases.

In 1950 Eliel *et al.* reported a series of 32 cases of potassium deficiency, of whom all save one had been maintained for several days on intravenous fluids containing no potassium. More recently Lans *et al.* (1952) have reported on a series of 404 cases of hypokalæmia, of whom the very large majority had no potassium intake for several days, either following operation or before admission to hospital. During such a period of negligible or absent intake there is a constant loss of potassium from the kidneys, which do not conserve this ion as strictly as sodium or chloride. This loss is accentuated after operation by the characteristic potassium diuresis, which is without doubt a critical factor in the production of significant potassium deficits, as by depleting the potassium reserves it renders the body vulnerable to further losses. All of the 32 cases of potassium deficit reported on by Eliel *et al.* (1950) occurred following operation, and the importance of the urinary loss was underlined by Tarail and Elkinton (1949), who studied a series of cases of hypokalæmia and found that in nearly every case the urine loss was greater than the overt loss from the gastro-intestinal tract, despite the higher concentration of potassium in the latter.

Losses of potassium from the intestinal tract are, however, of great importance in the production of potassium deficiency, as the concentration of potassium in the alimentary secretions is higher than that in the plasma (Table IX), so that losses of up to 100 mEq per 24 hours can occur from this source. By themselves such losses rarely cause potassium deficiency, but occurring in a patient already depleted by urinary losses ("differential intracellular depletion") they may well prove decisive in producing a serious deficit ("extra-

cellular depletion"). In the series of Eliel *et al.* (1950), 23 out of the 32 cases had losses from the gastro-intestinal tract, whilst Lans *et al.* (1952) report that, of 241 patients with pre-operative potassium deficiency, all but 15 had a disease of the gastro-intestinal tract, and all but 9 had an intra-abdominal disorder. Of the 69 cases of persistent post-operative hypokalæmia reported on by these latter authors, all had undergone abdominal operations. This triad of events, namely deficient intake, increased urinary loss, and loss from the gastro-intestinal tract, is of supreme importance in the development of potassium deficiency, and, indeed, the recognition of their occurrence is one of the aids to diagnosis.

The Clinical Picture of Potassium Deficiency

The clinical picture of potassium deficiency is not clear-cut, though with experience its presence can usually be suspected if not confidently diagnosed. The outstanding features are lethargy, apathy, anorexia and nausea, muscular hypotonicity, often abdominal distension, and sometimes dependent œdema (see Appendix B, Case 6). Peripheral vasodilatation is often present, sometimes sufficient to cause palpable pulsation in the pulps of the fingers, and in severe cases disturbances of cardiac rhythm may occur. As seen typically the patient lies listlessly in bed, refusing all fluids and moving his limbs little, if at all In the series of cases reported by Lans *et al.* (1952) this muscular hypotonus was the outstanding clinical feature, and in 6 cases there was actual muscular paralysis. Paralysis has been reported previously as occurring with hypokalæmia, but is distinctly uncommon, it is peculiar in that it usually affects the legs, rarely if ever the muscles supplied by the cranial nerves, and the reflexes are often present in the paralysed limbs. We have ourselves seen a severe state of hypokalæmia complicating bilateral ureterosigmoidostomy, with a plasma K level of 2·0 mEq/l., in which there was complete paralysis of the legs, severe weakness of the arms, but no interference with the facial musculature.

Abdominal distension, amounting to chronic ileus, is a common feature, so much so that the failure of paralytic ileus to respond to standard treatment strongly suggests the presence of hypokalæmia Eliel *et al.* (1950) reported that abdominal distension was common in their cases, and Lans *et al.* (1952) stated that it was marked in 31 of their cases. Indeed, the association between abdominal distension with chronic ileus and hypokalæmia is so marked that it has been suggested by several authors that potassium deficiency is the cause of paralytic ileus. This theory was put forward by Streeten and Ward-McQuaid (1952), who reported on 13 cases of paralytic ileus they had studied. They found that during the ileus there was an excess urinary loss of potassium, with lower-

ing of the plasma potassium concentration, as well as that of sodium and chloride. They also found that during ileus there was a retention of sodium and chloride, but that on recovery there was a loss of salt with retention of potassium. On the basis of these findings they suggested that paralytic ileus is due to potassium deficiency, which in its turn is caused by prolonged adrenocortical activity in the presence of an inadequate potassium intake. Streeten and Vaughan Williams (1952) investigated the same point experimentally in dogs. By means of intraperitoneal infusions the animals were depleted of sodium chloride, leading to a shift of potassium out of the cells with production of a severe intracellular potassium depletion, and under these distinctly artificial circumstances they found marked depression of gut movement, but it is to be noted that in these animals there was a considerable rise in the plasma potassium level, as opposed to the fall usually found in clinical cases of paralytic ileus.

There can be little doubt that potassium depletion depresses gut motility, and similarly that in many cases of paralytic ileus the administration of potassium is essential to, or at least greatly hastens, recovery. But it would appear to be overstating the case to say that potassium depletion is the cause of paralytic ileus. The weight of evidence suggests that the main factors in the development of this condition are the swallowing of air into an intestinal tract whose motility has been inhibited, usually by an abdominal operation. Potassium depletion is of importance in that, by further depressing intestinal motility, it may precipitate frank ileus in border-line cases, or exacerbate and prolong the established condition. In this connection it should not be overlooked that for many years variations in the plasma concentration of sodium have been known to influence gut motility (Orr *et al*, 1931; Perazzo, 1937). It seems likely that in paralytic ileus the sodium and potassium losses into and from the intestinal tract, by further depressing gut motility, set up a vicious circle which can only be broken by replacing these electrolyte losses.

Potassium deficiency is almost invariably accompanied by a degree of alkalosis,* and in some cases this is marked, giving rise to the condition of "hypochloræmic alkalosis". Attention was directed to this condition by Randall *et al.* (1949), who reported 3 cases, all of whom had complicated dehydration disturbances after operation, treated over a period of several days by "generous" transfusions of saline and dextrose; all showed drowsiness, chronic ileus, oliguria, peripheral œdema, hæmoconcentration, azotæmia,

* The [illegible] y
outl[illegible] e
ques[illegible] -
cuss[illegible] Black (1953).

a lowered plasma Cl but normal Na concentration, and a low plasma potassium; all 3 recovered rapidly when given potassium. Since that time the condition has become well recognised, and many cases reported: thus Lans *et al.* (1952) report that in 64 of their cases of potassium deficiency there was an alkalosis with an alkali reserve in excess of 33 mEq/l., and that in 41 of these there was an associated hypochloræmia. Moore and Ball (1952) discuss the condition in detail, and consider that its development depends on four events: (1) "Differential intracellular depletion", which renders the patient vulnerable to hypokalæmia, followed by (2) Metabolic alkalosis, usually produced by excess chloride loss from the gastro-intestinal tract. (3) Stress with adrenocortical release causes further potassium deficit, and often (4) a retention of sodium, exacerbating the alkalosis. Typically this series of events is seen in patients with pyloric stenosis who have been vomiting for some time (dehydration; metabolic alkalosis; extracellular K depletion), who are prepared for surgery with intravenous saline but no potassium, and then operated upon (sodium retention, with further K losses). The condition may, however, arise under other circumstances, and its recognition is important, as saline and glucose infusions only exacerbate the disturbance, which responds rapidly to the administration of potassium.

The Diagnosis of Potassium Deficiency

The diagnosis of potassium deficiency depends in the first place on bearing in mind the circumstances under which it is likely to occur, namely an inadequate intake, with increased urinary losses, and/or gastro-intestinal losses. In most cases clinical examination will show some, if not all, of the features already discussed. The diagnosis should always be suspected in the presence of prolonged ileus, or in cases with an acid-base disturbance, particularly an alkalosis, which do not respond to normal treatment. The diagnosis may be confirmed either by a serum potassium estimation, or by the performance of an electrocardiogram. The normal serum potassium level is 3·9–5·2 mEq/l.; a figure below 4·0 mEq/l. is suggestive of potassium deficiency, and one below 3·7 mEq/l. diagnostic, but it must not be forgotten that the condition can occur with a normal plasma level, particularly when associated with marked dehydration. The E.C.G. changes are particularly useful in that there are few hospitals in which this examination cannot readily be performed, and the result is quickly available. In addition, the E.C.G. curve will show abnormalities in many of those cases of potassium deficiency with a normal plasma potassium level. The interpretation of the E.C.G. curves is essentially a problem for the expert, but the changes are often marked, and the main abnormalities to be looked for are

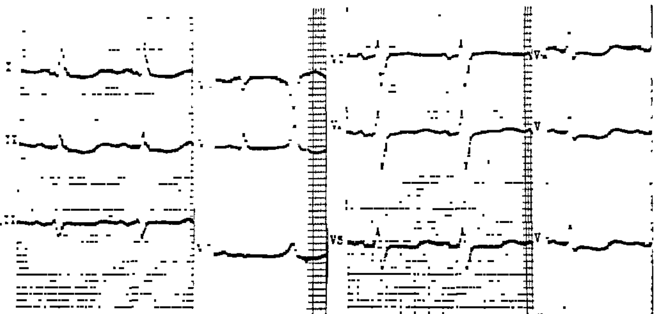


Fig. 26.—Electrocardiogram in potassium deficiency. Characteristic "sagging" depression of ST segment is seen in leads I, II, V4–6. T waves cannot be identified, being fused with U waves which are prominent in all leads. The rhythm was irregular from frequent premature ventricular beats, one of which is recorded in the unipolar limb leads (VR, VL, VF).

depression of the ST segment, lowering, widening or inversion of the T waves, prolongation of the QT interval, and the appearance of U waves (Fig. 26). (See Somerville, 1951, and Lewis, Somerville and Le Quesne, 1954, for full description of E.C.G. changes and references to literature.)

Treatment of Potassium Deficiency

In the treatment of potassium deficiency, a suitable salt should be given by mouth whenever possible, as there are risks attached to intravenous administration. In most cases the deficit is between 100–200 mEq, and replacement should not be carried out at a rate in excess of 100 mEq per 24 hours. For oral use potassium chloride is a suitable and convenient salt, and 100 mEq in 24 hours is provided by 30 gr. (2 gm.) 6-hourly, which may be given in fruit drinks. Alternatively, an equivalent amount of potassium citrate may be given—that is, 40 gr. 6-hourly.

When potassium has to be given intravenously, certain definite precautions should be observed, otherwise the plasma potassium concentration may reach dangerous levels, and cause serious derangements of cardiac function (see below). In the first place, potassium should not be given intravenously unless there is a good urine output: accordingly, it should not be given to a seriously depleted patient until the salt and water deficit has been at least partially replaced and urine output restored. Secondly, it should only be given with great care to patients known to have depressed renal function. Thirdly, the concentration of potassium in the fluid used should not exceed 40 mEq/l., the rate of administration should not exceed 20 mEq/hr., and the total dose should not exceed 100 mEq in a 24-hour period. Provided these precautions are taken and that the progress of the patient is checked by daily plasma potassium estimations and/or an E.C.G. record, no harm need be anticipated. It may well be that hitherto the dangers of giving potassium intravenously have been exaggerated, and that in special circumstances it may be necessary to give potassium in excess of the quantity and rate outlined above. But it is wise not to exceed these unless obligatory, and until considerable experience in the use of potassium solutions intravenously has been obtained, when the daily checks can also be relaxed. Especial care should, however, be taken if for any reason it is found necessary to give intravenous potassium to a patient with a low urine output. Under such circumstances a dangerous rise in the plasma potassium level may easily be caused. The earliest signs of cardiac derangement are seen when the plasma potassium concentration rises above 7 0 mEq/l., serious manifestations develop at 9–11 mEq/l., and death from cardiac arrest occurs at a level of 14–15 mEq/l. (see Somerville, 1951).

A large variety of different fluids have been suggested and used for giving potassium intravenously, most of which are essentially similar. Although experience shows that with care solutions of a concentration of up to 80 mEq K per litre can be used, this is not advisable. With solutions of this strength there is a definite risk of causing a dangerous rise in the plasma potassium level if the fluid is given too quickly, and in addition they cause considerable pain whilst running in. For these reasons the solutions used should not exceed a strength of 40 mEq/l., and some authorities advise even weaker mixtures. We have used predominantly the following two solutions:

(1) KCl 3·0 gm/l. (K 40 mEq/l.: Cl 40 mEq/l.);

(2) KCl 3·0 gm/l.: NaCl 2·25 gm/l. (K 40 mEq/l.: Na 40 mEq/l.: Cl 80 mEq/l.).

Only rarely have we given over 2 litres of either fluid in 24 hours, and care has been taken to observe the precautions mentioned, but on no occasion have we seen any dangerous effects during the four years we have been using these solutions. The mixed saline and potassium solution is of particular use in the treatment of observed losses, and when these exceed 2 litres per day the replacement of 1 litre with this mixed solution, instead of with normal saline, is of great value. On occasions, pain may occur in the vein when potassium is being given. This can usually be relieved by slowing the rate of the drip, or diluting the potassium solution with the succeeding bottle of saline or glucose. If both these methods fail, an injection of 2 ml. of 2 per cent. procaine into the drip near the needle is frequently successful.

Two further points in connection with potassium administration need mention. First, for reasons which are not understood, the administration of potassium to a depleted patient is followed by a fall in the urine output of potassium (Appendix B, Case 4), not a rise as would be expected; if for any reason the urine potassium figures are being closely followed, this may lead to a mistake in treatment unless its significance is recognised. Secondly, in cases of marked depletion with contraction of the extracellular space, the plasma potassium concentration may well be normal, thus masking a large deficit However, when the water and salt deficit is replaced, a rapid fall in the potassium concentration will occur and a serious degree of hypokalæmia may be precipitated, partly by the dilution of the extracellular potassium, and partly by the increased loss caused by saline transfusions. Accordingly, this possibility should be carefully borne in mind, and directly the urine flow has been re-established, potassium should be given (Appendix B, Cases 3 and 4).

In a previous chapter the question of the routine use of potassium solutions intravenously has already been discussed. Bearing in mind

that one of the main factors in the production of potassium deficiency is the negative balance which develops during a period of deficient or absent intake, it is illogical to maintain a patient for many days on an intravenous intake of glucose and saline solutions only and thus allow a potentially serious deficit to accrue. There is no evidence that an absent intake for 24–48 hours is harmful, but if intravenous fluids must be given for longer than this it is wise to add potassium to the basic intake. In our experience this measure is quite safe, practically abolishes the development of potassium depletion after operation, and materially diminishes the incidence of established ileus.

RENAL FAILURE: ANURIA

In the maintenance of water and electrolyte balance the kidneys exercise a control the accuracy of which it is impossible for the physician or surgeon to equal, and the treatment of most derangements of fluid balance depends ultimately for success on the ability of the kidneys to even out the differences between intake and output, by excreting or retaining more or less of the various ions or of water. This reliance on kidney function has been emphasised repeatedly in discussing the various problems of dehydration, and it is obvious that if there is any significant depression of this function the occurrence of serious disturbances in water or electrolyte balance will lead to problems of great complexity. In theory it should be possible to handle such a situation by accurately measuring all deficits and losses and then replacing them exactly, but in practice this is not feasible, and experience shows that unless there is a reasonable flexibility of renal function, a disturbance of any magnitude will inevitably lead to an uncontrollable biochemical disorder and death.

Fortunately, severe degrees of renal failure are not common in surgical patients, but cases with minor degrees of depression of function are frequently seen. The essential feature in the handling of these cases is to give plenty of water, since failure of urinary dilution occurs at a much later stage than failure of concentration. Reference to Fig. 4 shows that as the concentrating power of the kidney diminishes, so the minimal obligatory urine volume increases; if sufficient water is not given to allow for this increased urine volume, dehydration will occur, leading to further depression of renal function and eventually to uræmia. In the same way the other functions of the kidney are depressed, so that it is essential for these patients to have a generous water intake, with a normal electrolyte intake, so that the kidneys can work within the limits of their diminished capacities.

Apart from these chronic disturbances, by far the most important derangement of kidney function seen in surgical patients is acute tubular necrosis ("lower nephron nephrosis"), leading to oliguria and sometimes complete anuria. This is a highly complex subject and the following is only an outline of the principles by which such cases should be treated Excellent reviews of the condition are given by Bull (1952 and 1955) and Swann and Merrill (1953), and the aspects of the subject of importance in surgical practice are admirably discussed by Kolff (1955). The essential lesion in such cases is a necrosis of the tubules, usually as a result of a severe fall in blood pressure

or a mismatched blood transfusion, and most cases occur following an operation or after an abortion. Viewed functionally, this necrosis results in a complete inability of the kidney to make any adjustments in urine output to meet changes in water and electrolyte intake. Initially, the tubules appear to reabsorb almost all the glomerular filtrate so that there is oliguria, with retention of the products of tissue breakdown. During the period of recovery the tubules, contrariwise, reabsorb little or none of the filtrate so that there is then polyuria with a urine of fixed concentration (specific gravity about 1010), closely resembling extracellular fluid. Later the tubules gradually recover their full flexibility and concentrating powers, and become once again able to secrete a urine adapted to the intake and the needs of the patient.

Spontaneous recovery will usually take place in 1–4 weeks, if the patient does not die during this period from disturbances of water balance or from intoxication by the products of tissue breakdown. The retention of urea itself is not harmful, but many of the other products of katabolism are highly toxic, and in many cases death is probably due to potassium poisoning (Strauss, 1948). Unless tissue breakdown is reduced to a minimum this is likely to occur on about the 10th–12th day, when the plasma potassium level has risen to 14–16 mEq/l.

It is important that the diagnosis should be made promptly, as a successful outcome is largely dependent upon the institution of correct treatment in the early stages. Inevitably, the presenting manifestation is oliguria or anuria, and in the majority of cases this will have been preceded by the precipitating factor, usually a severe fall in blood pressure, as a result of hæmorrhage or shock, or a mismatched blood transfusion, though in occasional cases (such as the rare hepato-renal syndrome) such a factor is not obvious. Complete anuria only occurs in severe cases. In the less severe, such sparse urine as is formed will usually contain albumin and blood and quite early in the condition will show a fixed specific gravity around 1010. Within 24–48 hours of the onset the blood urea and serum potassium figures will rise, and if, during this period, a considerable amount of water has been given the serum sodium and chloride figures may well be low. In most cases the diagnosis is obvious, but on occasions it may be necessary to distinguish the condition from oliguria due to other causes, the most important in surgical practice being a deficient intake or unreplaced abnormal losses, and this problem is discussed in a previous section (p. 53).

Bearing in mind that spontaneous recovery usually occurs, treatment should be directed towards two main ends, namely the reduction of tissue breakdown to a minimum so as to allow the maximum time for this recovery and, secondly, the provision of a water and

salt intake adjusted as closely as possible to the output. These patients cannot be made to secrete urine by increasing the water intake, and any attempt to force a urine flow by such means can only lead to water retention. This danger of over-administration applies even more strongly to salt, for, whilst excess water can over a period of time be removed as insensible loss, there is no such safety valve for salt. There is no evidence that renal decapsulation or renal periarterial sympathectomy have any influence on the course of the disease, and the administration of sodium sulphate is positively harmful during the period of anuria, since the retention of these ions increases the abnormality of the extracellular fluid.

These therapeutic requirements are met by providing only sufficient water to replace losses from the skin and lungs, and in addition a full calorie intake in the form of fat and carbohydrate (Bull, Joekes and Lowe, 1949; 1950). During the period of anuria the daily requirements of these patients are therefore (*a*) 1000 ml. of water, (*b*) no salt, unless there are abnormal losses, (*c*) 2500 cals. provided by 400 gm. of glucose and 100 ml. of arachis oil emulsified with acacia. This mixture of oil and glucose in water is so thick and unpalatable that it must be given down a naso-gastric tube, but even then it may cause vomiting. If vomiting does occur, the vomit should be collected, filtered through gauze and returned down the stomach tube. In view of this tendency to produce vomiting (or sometimes diarrhœa) and to allow for the endogenous water production, Bull (1955) recommends that during the period of anuria the oil should be omitted and that the water intake should be restricted to 600–800 ml. per 24 hours, and it is certainly wise to err on the side of giving slightly too little rather than too much water. This treatment prevents waterlogging, and reduces tissue breakdown to a minimum, so that the blood urea and plasma potassium only rise by, respectively, 12 mgm. per cent. and 0·32 mEq/l. per day (Strauss, 1948), thus greatly prolonging the time in which recovery may occur. It must be emphasised again that a rising blood urea is not in itself harmful, but its estimation acts as a most useful index of the progress of the disease.

Once the urine flow returns and polyuria develops, the intake should be increased by an equal amount of water, which may be drunk. In addition, the salt content of the urine should be estimated and an equal amount added to the intake. During this period of polyuria there is also a considerable potassium loss in the urine, with a risk of the patient developing hypokalæmia, so that the plasma potassium concentration should be estimated at frequent intervals and a suitable potassium salt given by mouth if there is evidence of deficiency. Replacement on these lines should continue until the renal function is reasonably flexible, when a normal intake

can then be given safely. Careful treatment during this period of polyuria is as important as during the stage of oliguria or anuria, and the patient's water and electrolyte intake should be controlled with care until observations (e.g. of the urine specific gravity) show that the kidneys are once again able to adapt themselves to altering loads.

Patients with acute tubular necrosis are particularly liable to develop, and to succumb to, acute infective processes. They should, therefore, be nursed with great care, and some authorities (Bull, 1955) recommend the use of modified barrier nursing. It is a wise precaution to give ½–1·0 million units of crystalline penicillin daily, but owing to the low or absent urine output other antibiotics should only be given if absolutely indicated, and then in modified dosage, and the sulphonamides should, of course, be avoided. Anæmia also has a very deleterious influence on these patients and retards recovery. Accordingly, the hæmoglobin should be checked at frequent intervals, and if it falls a blood transfusion given, preferably using packed red cells.

If abnormal losses occur during the course of treatment, they must be accurately replaced. Thus, if the patient is sick, the vomit is returned down the Ryle's tube. If the loss is not replaceable in this way, it should be measured, its salt content estimated, and appropriate additions made to the feed. A very useful guide to the accuracy of such replacement is given by daily weighing of the patient, as the provision of adequate calories means that any sudden alterations in weight can only be due to alterations in the patient's state of hydration. A very difficult situation arises if this treatment has to be given to a patient who is initially deficient in salt and water; in such cases replenishment must be carried out with the utmost discretion, care being taken to under- rather than over-estimate the deficit. A further problem arises if, owing to the conditions of the case, it is not possible to give fluids by mouth. There is no difficulty in giving 600–1000 ml. of water daily by intravenous infusion, but it is more difficult to provide a sufficient calorie intake. However, an adequate calorie intake can be provided by using hypertonic glucose solutions. Such solutions are very liable to cause thrombosis in small peripheral veins, and under these circumstances a caval drip should be set up (see later section). Using this technique, glucose solutions up to a strength of 50 per cent. can be administered, and 50 per cent. glucose in water is probably the fluid of choice in this situation.

If recovery is greatly delayed, it may be necessary to remove toxic products from the body by the use of an artificial kidney, peritoneal or intestinal dialysis, exchange-transfusion or (to remove potassium) the administration of ion-exchange resins. These forms of treatment

demand the attention of an expert physician or biochemist, and are not within the compass of most surgical departments. However, this emphasises the importance of instituting treatment as early as possible in cases of anuria, so as to allow the maximal time for recovery. In this connection, it must be emphasised again that complete anuria is not an essential part of the syndrome—in fact, it is uncommon. Severe oliguria (200–300 ml. daily) is usual, but the treatment required is the same (Appendix B, Case 7), and is just as urgent. *In all patients with oliguria it is therefore of the utmost* importance to decide the cause (Appendix B, Case 8), so that if acute tubular necrosis has occurred, treatment may be started promptly.

EFFECTS OF EXCESS WATER AND SALT

It has already been stressed that the oral and intravenous routes are not *simple alternative methods of giving* fluid, and that the intravenous route should only be used when definitely indicated. One of the great advantages of giving fluid by mouth is that it is very difficult to overload a patient in this way, as before this can be achieved the patient will start refusing further drinks. However, when the intravenous route is used, the safeguard provided by the patient's objections is removed, and the danger of overloading is appreciable.

Pure water intoxication is rarely seen, as a considerable excess must be given to cause symptoms, and this is likely to occur only if excessive administration is continued in the presence of obvious kidney failure. There is, however, a real danger of overloading a patient with water during the first 18–36 hours after operation, as during that time the kidneys, though not in failure, have a specific inability to excrete water. As a result, excessively rapid administration of either intravenous glucose or rectal fluids, e.g. tap water, during this period can easily lead to dangerous overloading, with the production of water intoxication. This post-operative complication was first described by Helwig, Schutz and Curry in 1935, who reported on a fatal case dying in convulsions 41 hours after a cholecystectomy, during which time the patient had received 9 litres of tap water by proctoclysis. Three years later Helwig, Schutz and Kuhn (1938) reported a further case, occurring in a woman after a panhysterectomy. In this latter case the first symptoms developed 36 hours after operation, at which time the patient had received 7000 ml. of tap water by rectal tube, and 12 hours later, after the administration of a further 1250 ml. of water by the same route, the first convulsion occurred: at this point 5 per cent. saline was given intravenously, with prompt relief of symptoms and recovery of the patient. Zimmerman and Wangensteen (1952) reported 17 cases of post-operative water intoxication, and their paper describes the condition in full. Sixteen of their cases occurred within 48 hours of operation, and the majority developed in the period 12–36 hours after operation. Unconsciousness or coma were the predominating signs in the majority of their cases, 3 of which ended fatally. All showed marked extracellular hypotonicity, with distinct falls in the plasma sodium and chloride concentrations, and in nearly all the

cases the body-weight was higher than pre-operatively, in several instances the gain being 2–4 Kg. (4·4–8·8 lb.). Most of these cases had received excessive amounts of salt-free fluids prior to the development of the condition, and all those treated by prompt administration of sodium chloride with restriction of water intake recovered rapidly. Recently Wynn and Rob (1954) have reported further cases, two again due to the excessive administration of rectal fluids, and they discuss the condition in detail.

In their discussion of the condition Zimmerman and Wangensteen (1952) suggested that it is due to an inadequate or abnormal adrenocortical response to operation. But, bearing in mind that it almost invariably occurs within 48 hours of operation, that it is accompanied by distinct extracellular hypotonicity and that it is rapidly cured by restriction of water intake and the administration of salt, it seems most probable that it is, in fact, an extreme manifestation of the post-operative impairment of water excretion, and this is borne out by detailed observations which we were able to make on a case with this condition (Le Quesne, 1954). The patient, a slight woman of 67 years weighing only 35 Kg., underwent a resection of the rectosigmoid with end-to-end anastomosis. Her recovery was uneventful until some 48 hours after operation, when she became very restless and agitated, with constant nausea and retching, though she was not actually vomiting. On examination, her abdomen was flat and soft with no sign of peritonitis or ileus; her blood pressure was not significantly different from the pre-operative level, but the peripheral veins were full, with slight but definite jugular filling, and there was minimal sacral œdema. Examination of the venous blood gave the following results: Hb 77 per cent.: plasma Na 122 mEq/l. plasma Cl 78 mEq/l. Up to that time (48 hours post-operatively) the patient had received intravenously 500 ml. of citrated blood, together with 6000 ml. of water and 120 mEq NaCl, during which time she had excreted 1870 ml. of urine, the specific gravity being still 1029 at the end of this period. During the next 24 hours little change in the condition occurred, but at the end of this period there was a profuse diuresis, accompanied by a dramatic clinical improvement of the patient, a drop of nearly 2 Kg. (4·4 lb.) in body weight, and a rise of the blood estimations to the following figures: Hb 89 per cent.: plasma Na 147 mEq/l.: plasma Cl 91 mEq/l. Figure 27, constructed from observations made on 4-hourly urine specimens collected by urethral catheter, shows the changes in urine output and specific gravity for 96 hours after operation. These changes are precisely similar to those of post-operative impairment of water excretion, save for the fact that they continued for 72 hours after operation instead of for the usual 24–48 hours. The evidence that these changes are due to a secretion of a

has already been discussed, and these observations strongly suggest that in fact post-operative water intoxication is due either to an exceptionally prolonged secretion of this hormone (as in this case), or to excessive administration of electrolyte-free fluid during a normally prolonged episode of primary water retention. Either of these situations will lead to an excessive retention of water within the body, causing extracellular hypotonicity and so intracellular overhydration, this latter probably being the essential cause of the main symptoms.

As is apparent from the description above, the initial symptoms of water intoxication are nausea, retching, and restlessness, going on later to coma and convulsions. Physical signs are minimal, the pulse

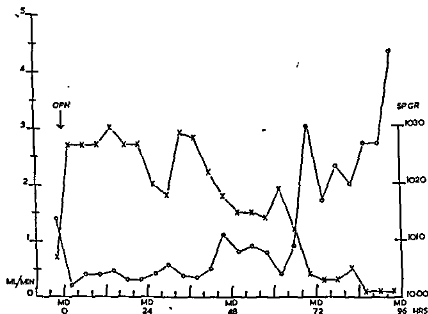


FIG. 27.—Data from a case undergoing sigmoid colectomy, who developed signs of water intoxication 48 hours after operation (see text). The operation is marked by the arrow. The graphs record the minute volume of urine (circles) and urine specific gravity (crosses) of successive 4-hourly urine specimens. In this case the post-operative impairment of water excretion lasted 72 hours (*cf.* Figs. 9 and 17).

being full, skin elasticity normal, and œdema is rarely present. Estimation of the plasma electrolyte concentrations will show hypotonicity *of a varying degree*, depending on the extent of the overloading with water, and this will also be reflected in the level of hæmoglobin concentration. If, as in most cases in surgical practice, the condition develops shortly after operation the urine output will be low, with a raised specific gravity, and containing no abnormal constituents. Confirmation of the diagnosis can be obtained by a

consideration of the intake and output figures for the preceding 24–48 hours and in assessing the significance of these figures care must be taken not to overlook the insensible loss. Once the diagnosis has been made further water intake must be stopped, and in mild cases nothing further is required, but in severe cases with marked hypotonicity the prompt intravenous administration of hypertonic saline is essential. Wynn and Robb (1954) used 6 per cent. saline in the treatment of their cases, and recommend that it should be given in amounts of 100 ml. at a time, injected over a period of a few minutes, and repeated if necessary some 30 minutes later: usually 200–400 ml. are sufficient to relieve the coma and convulsions, and thereafter water restriction should be continued until recovery is complete.

Overloading with sodium chloride, that is normal saline, is both more readily produced than pure water overloading, and a danger that is more commonly recognised. Overloading with salt increases the extracellular space, affecting both its interstitial and intravascular compartments. This will lead to many consequences, in particular œdema at the lung bases and in wounds, increasing venous pressure and, finally, obvious anasarca, with dependent œdema, ascites and heart failure. Examination of the blood will show hæmodilution, with lowered hæmoglobin and plasma protein concentrations, and possibly raised serum sodium and chloride concentrations. Again, because of the impaired salt excretion at this time, there is an especial danger of this type of overloading following operation, and it must be emphasised again that during this period the standard basic intake (p. 48) should not be increased without good evidence of abnormal losses.

This danger of giving excessive quantities of saline after an operation has been clearly recognised for many years, and in their paper describing post-operative salt intolerance Coller and his associates (1945) described 5 cases made œdematous in this way. The danger was well illustrated in the case of a man treated by us some years ago, who developed severe paralytic ileus after a gut resection for strangulated femoral hernia. This patient received as his basic salt intake per 24 hours, 2 bottles of normal saline, i e. 1 litre containing 9 gm. of sodium chloride; over and above that the abnormal losses from the alimentary tract were replaced volume-for-volume with normal saline. For a period of 2–3 days after operation these losses, in the form of aspirate from a Ryle's tube, amounted to around 5 litres per 24 hours, so that this man was receiving in excess of 5 litres of normal saline (750 mEq Na) intravenously per 24 hours. Figure 28 shows the sodium balance from this patient, and it will be seen that for 5 days after operation there was a marked retention of sodium, so much so that sacral and ankle œdema developed on

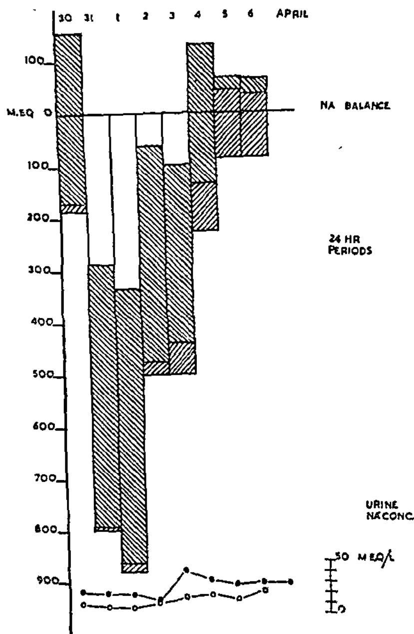


FIG. 28—Sodium balance and urine electrolyte concentrations from a case developing paralytic ileus following gut resection for a strangulated femoral hernia Operation was performed a few hours before the start of the graph, and ileus developed during the second 24-hour period illustrated, the intestinal aspirate continuing copious for the next few days. Note the large sodium retention in the presence of very low concentrations of sodium (dots) and chloride (circles) in the urine. In this instance the lowered urine electrolyte concentrations are a manifestation, not of salt depletion, but of the body's impaired ability to excrete salt after operation.

the fourth day. The same figure also shows that throughout the period of retention there was a very low concentration of sodium *in the urine, demonstrating that the retention was not due to the* fact that there was an insufficient volume of urine in which to excrete the excess sodium, but was in fact due to the post-operative impairment of salt excretion which in its turn led to œdema formation. In the same figure (Fig. 27) the daily urine chloride concentrations are also plotted, showing that throughout the period of retention the amount of chloride in the urine was negligible. It is essential to note that under these circumstances this very low urine chloride concentration is not, as it normally is, indicative of a shortage of salt, but is simply a manifestation of the body's inability to excrete salt during the post-operative period. Such a dramatic discrepancy between the apparent significance of the urine chloride concentration and the actual salt balance of the patient illustrates clearly the care with which this simple test must be interpreted after an operation.

Treatment obviously consists in withholding further salt whilst providing a reasonable water intake, and in gross examples a venesection may be required. It must not be overlooked that such a condition of sodium chloride excess can exist in the presence of a water deficit, causing oliguria and thirst; treatment in this situation is in no wise different, except that potassium will probably be required when a reasonable urine output has been established. The dangers of sodium chloride retention are considerably increased if there is a coincident protein deficiency with hypoproteinæmia. This will cause an abnormal distribution of water in the extracellular space, so that œdema will occur both more readily and to a greater extent, and a comparatively slight excess of salt can easily cause harmful œdema at suture lines and other vulnerable sites, leading to tiresome if not serious complications. In any patient in whom an intestinal anastomosis is unduly slow to start working or who develops unexpected pulmonary complications, the fluid intake should be critically examined in relation to this possibility, and if necessary a plasma transfusion given to restore normal distribution within the extracellular space.

NITROGEN AND CALORIE PROBLEMS

THE maintenance of water and electrolyte balance is only one part of the more general problem of the maintenance of nutritional equilibrium in the surgical patient. In that disturbances of fluid balance are frequently acute in onset and require prompt recognition and treatment, they undoubtedly demand prior consideration. By contrast, disturbances of protein (nitrogen) and calorie intake are commonly insidious in onset and often far from obvious in their effects. Nonetheless, from time to time such disturbances give rise to serious problems and require more conscious attention than is customarily paid to them. A full consideration of nutritional problems in surgical practice is beyond the scope of this monograph, but the main features demand some mention.

In general, serious nutritional problems most commonly arise in the period after operation. Any operation is necessarily followed by a breakdown of protein (the katabolic response), resulting in an increased urinary excretion of nitrogen. This nitrogen diuresis is an integral part of the metabolic response and in general its size and duration vary with the severity of the operation. After an operation of the magnitude of a partial gastrectomy the increased nitrogen excretion lasts some 24–48 hours (Fig. 29), but greater losses than this occur after very major operations. In the majority of patients this increased nitrogen loss occurs at a time when their protein and calorie intake is diminished, especially if the patient is receiving intravenous fluids, and accordingly results in a negative nitrogen balance over this period. Further, it appears that it is probably impossible to reverse this negative balance during the first few days after major surgery (Ariel *et al.*, 1943), though it is to be noted that there are no observations of the effect of really high calorie and protein intakes maintained throughout the period of operation and for several days thereafter. However, just as this katabolic process is an integral part of the metabolic process, so necessarily a subsequent period of positive nitrogen balance with a laying down of new tissue is an essential part of the full recovery from a surgical operation. Moore (1953), discussing the bodily changes in surgical convalescence, breaks down the metabolic response to trauma into four phases. The first two phases essentially cover the nitrogen diuresis and the water and electrolyte changes described previously; the latter two, named by him the S[illegible] Phase and the Fat Gain Phase, [illegible] Moore describes the Spontaneous [illegible] some 7–10 days

after major abdominal surgery, at a time when full oral feeding is re-established, and its outstanding feature is the replacement of the body protein broken down during the katabolic stage, a process which takes place before the final recuperative episode, the restoration of the fat depots (the Fat Gain Phase).

These recuperative stages are an essential part of the full recovery from a surgical operation, and should be regarded as part of the

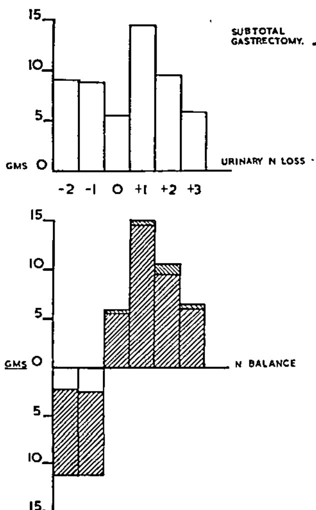


FIG. 29.—Nitrogen balance and urinary nitrogen excretion from a patient undergoing sub-total gastrectomy. Before operation this man was on a continuous, intra-gastric drip feed, and was in positive nitrogen balance On the day of operation itself (O) the urinary nitrogen loss was not increased, owing to a very low urine output, but for the next 48 hours there was an obvious nitrogen diuresis Occurring at a time when the patient was on intravenous fluids and received no protein, this increased urinary loss caused a markedly negative nitrogen balance over these days.

total metabolic response of a patient to a surgical operation. In the great majority of patients this anabolic process takes place quite spontaneously as a full dietary intake is regained. However, in a minority of patients, for various reasons, there may be a failure or impairment of these restorative phases, leading to a slowing down or halt in recovery from operation. Such a situation most often arises in patients who have lost considerable weight before operation and who have to undergo major surgical treatment, perhaps involving more than one operation or a stormy post-operative course, and particularly if they are left with large granulating wounds healing by secondary intention. A typical example of such a situation is the extensively burnt patient with large septic areas requiring frequent dressings and skin-grafting operations. Such patients require a high protein and calorie intake to heal their wounds, to replace the extensive losses as the result of the katabolic response and losses from the wounds, and to restore their general well-being, but often they are unable or too exhausted to take such an intake in the usual way. This failure to eat simply exacerbates the nutritional deficit, making the patient feel yet weaker and so setting up a vicious circle. In these patients the negative nitrogen balance is not, as immediately after operation, an inevitable metabolic response: it is rather a potentially lethal drain, and its reversal is essential to their recovery (Sutherland, 1954).

Before considering the management of such a situation it is necessary to stress some of the basic facts concerning nutritional equilibrium. The body only contains small carbohydrate stores, in the form of liver glycogen; in starvation these stores are soon exhausted, and then to provide the necessary calories the body burns fat and protein in a ratio approximately of 2·5:1. The requisite fat is readily available in the fat depots and can be consumed without any deleterious effects, but there are little or no reserves of protein, so that this process necessarily involves the destruction of body tissue, which takes place at the rate of about 70 gm. of protein per 24 hours. If such a patient is given fat and carbohydrate in sufficient quantities to satisfy his calorie requirements this rate of protein breakdown is greatly reduced, but there will still be a minimum breakdown, due to "wear and tear" of about 6 gm. of protein per 24 hours. If a patient is to restore a protein deficit which has arisen in this way he must be supplied not only with protein but also with sufficient fat and carbohydrate to meet his calorie requirements, otherwise the protein intake will be used, not for its unique tissue-forming properties, but as a source of calories, with excretion of the nitrogen as urea. Moore and Ball (1952) recommend that to allow of the laying down of new tissue the provision of 200 calories per gram of nitrogen (6·3 gm. protein) is the ideal ratio (Fig. 30) and it

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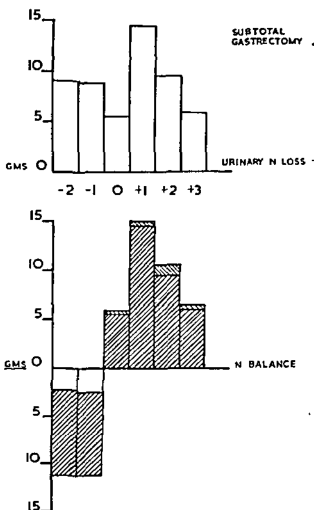


FIG 29—Nitrogen balance and urinary nitrogen excretion from a patient undergoing sub-total gastrectomy. Before operation this man was on a continuous, intra-gastric drip feed, and was in positive nitrogen balance. On the day of operation itself (O) the urinary nitrogen loss was not increased, owing to a very low urine output, but for the next 48 hours there was an obvious nitrogen diuresis. Occurring at a time when the patient was on intravenous fluids and received no protein, this increased urinary loss caused a markedly negative nitrogen balance over these days.

to making the diet attractive and palatable. The real problems arise in those few cases already mentioned in which severe losses have led to exhaustion and anorexia, in their turn aggravating the nutritional deficit. It must be clearly recognised that to replace their deficits such patients require an intake in excess of normal. In normal health a patient of average build consumes some 2000–2500 calories and 80 gm. of protein per 24 hours to maintain nutritional equilibrium, but these patients require an intake in the neighbourhood of 4000 calories and 100–150 gm. of protein. In some cases skilful dietetic care, with the administration between meals of high-calorie drinks fortified with protein digests, will provide an adequate intake, with reversal of the negative nitrogen balance and a steady return of normal appetite. Various oral fat emulsions, protein concentrates, etc., are now available to supplement such diets, but only too often, even when carefully disguised, chilled or flavoured, the patient finds them unpalatable and refuses to take them, and it may then be necessary to consider alternative methods of increasing the food intake.

In these circumstances we have found the use of tube-feeding by night very satisfactory. During the day the patient is encouraged to eat as much as possible without unduly bothering him. Each evening a fine tube is passed via the nose into the stomach and a slow drip feed given over 6–8 hours whilst the patient sleeps, the tube being removed each morning. This method has the advantage that the patient is troubled little during the day. The feed at night does not impair their appetite, but rather, with improving strength, enhances it. The exact composition of the feed can be varied to suit each case. No elaborate constituents are needed, and the help of expert dieticians is not required. Very satisfactory feeds can be made using skimmed milk powder, glucose and a simple fat emulsion, with any required electrolyte and vitamin additions, the whole being mixed in water with a simple domestic blender. The following formula, which can be augmented as required, is a satisfactory basic solution: skimmed milk powder 110 gm.: glucose 70 gm.: fat emulsion (50 per cent.) 150 ml.: NaCl 3 gm.: KCl 3·0 gm.: 1200 ml. water. This provides 40 gm. of protein, and 1400 calories. These quantities can often be increased with benefit and if desired the protein can be provided by Casilan (Glaxo) or some of the high-protein powders available, but there is no advantage in giving the protein in a predigested form.

In using such feeds two technical points are of importance. First the tube used should be as small and soft as possibl[illegible]

[illegible]

or a length of bicycle valve tubing. Secondly, to prevent nausea, or

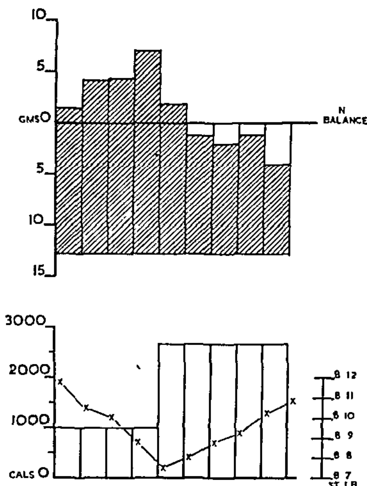


FIG 30.—Data showing the effect of calorie intake on nitrogen balance. Throughout the period of study this patient received a fixed protein intake (80 gms. protein: 12 8 gms. nitrogen) each 24 hours. During the first 4 days his total calorie intake was only 1,000 calories per 24 hours During the latter 5 days of the study, by the addition of fat and carbohydrate, the total calorie intake was increased to 2,680 calories per 24 hours. In the first period this man was in persistently negative nitrogen balance and lost weight steadily, but during the second period he was in positive nitrogen balance and gained weight. In the first period the intake provided 78 cals. per gram. of nitrogen, in the latter 208 calories

is in this provision of calories rather than protein that critical difficulties usually arise

In the first few days immediately after operation little attention need be paid to the provision of protein and calories, but towards the end of the first week and thereafter it becomes increasingly important to see that the patient's intake is adequate. As already mentioned, in the majority of cases this gives rise to no problems, or to none that are not soluble by a little cajolery and intelligent attention

do attempts to make them eat more cause a substantial increase in intake, but often, in fact, such attempts cause a revulsion to food and so do more harm than good (Co Tui *et al.*, 1944). Similarly, in most cases, tube feeding is impossible, but in some patients with partially obstructing neoplasms of the stomach, large colonic growths or other lesions causing considerable weight loss, it may be possible to institute satisfactory tube feeding or to get the patient to take adequate quantities of high-calorie liquid feeds. Despite the lack of proof of their benefit, if such treatment can be carried out satisfactorily, it is probably worthwhile to give these patients a high-calorie, high-protein intake for 7–14 days before operation, though in cancer patients operation should probably not be delayed longer.

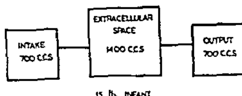
However, in many gravely depleted patients it is impossible to give an adequate intake by these methods. On many grounds the construction of a jejunostomy is undesirable, and is usually contra-indicated. Similarly, in the present state of knowledge, it is not possible to give these patients an adequate intake intravenously. An adequate protein intake can be given by administering a casein hydrolysate; such solutions contain all the essential amino-acids in a form available to the body (Elman, 1948), but they are of little value unless accompanied by an adequate calorie intake. Their value is increased by administering with them glucose in high concentrations (Ellison *et al.*, 1949), and further additional calories may be provided by adding alcohol to the intravenous fluids (Rice, 1949), but it is not possible to give a really adequate calorie intake unless a good proportion of it is provided by fat (1 gm. fat = 9 cals.: 1 gm. carbohydrate = 4 cals.). Various fat emulsions for intravenous use have been described and tried out clinically (Van Itallie *et al.*, 1952; Shafiroff and Mulholland, 1951), and more recently a new type has been reported on, with satisfactory results from animal administration (Payne *et al.*, 1956), but as yet no such emulsion has been found which is reliable enough for routine clinical usage and none is available commercially. In view of this difficulty in giving adequate calories intravenously, it is almost inevitable that if feeding must be by the intravenous route it will be accompanied by a steadily increasing nitrogen loss from the body (Fig. 40). Accordingly, if a patient cannot be fed in any way by mouth or tube feeding, once any water and electrolyte deficits have been replaced by intravenous fluids it is probably wise to proceed to operation forthwith, delaying attempts to replace solid nutritional losses until the post-operative, anabolic phase.

occasionally diarrhœa, the feed should be given at a constant, slow rate to prevent over-filling of the stomach. This can usually be achieved satisfactorily by the use of a screw clip on the tubing from the container and a drip chamber, but more certainty is obtained by using a pump, and Simpson and Dudley (1956) have recently described a simple gravity-operated pump for this purpose.

The benefit from this night, tube-feeding regime is often dramatic. With the establishment of a positive nitrogen balance not only do the patient's wounds begin to heal rapidly, but his vigour and appetite return and within a week or 10 days it may be possible to discontinue the treatment and return to normal oral feeding. Some writers have suggested that anabolic processes may be stimulated by giving various hormones, such as testosterone, but there is no conclusive evidence that they are effective, and their use is certainly harmful if it deflects attention away from the prime need to increase the protein and calorie intake. For reasons given below intravenous feeding is rarely of use in combating nutritional deficits in the post-operative patient.

Nutritional problems are also met with in the pre-operative period, particularly in patients with obstructive or ulcerating lesions in the upper alimentary tract. These patients, frequently requiring major surgical treatment, have often lost a considerable amount of weight due to deficient food intake and to vomiting, and present with multiple nutritional deficits; it seems obvious that they would withstand surgery better if they could be given a high-protein high-calorie intake for 10–14 days before operation, leading to gain in weight and general increase in vigour. There is no doubt that in such patients the metabolic response to surgery is less vigorous than in the normal (the depleted response, Moore and Ball, 1952), suggesting an impairment of their usual reactive processes. Further, there is no doubt that in these patients the anabolic process is of enhanced importance, and it is essential that as soon as possible after operation they obtain an intake sufficient to put them into positive nitrogen balance. Nonetheless, it is common surgical experience that depleted patients of this type, on the face of it poor risks, in fact withstand operation very well, provided there are no serious post-operative complications; and indeed there is no concrete evidence that the operative risk is diminished by a comparatively short period of pre-operative high-calorie feeding. As the result of such feeding, when possible, these depleted patients undoubtedly appear fitter and better able to withstand a major operation, but there is no factual proof of this impression.

In practice it is usually very difficult to give to such patients a high-calorie high-protein feed. In the great majority their primary lesion prevents them taking adequate food by mouth, and rarely



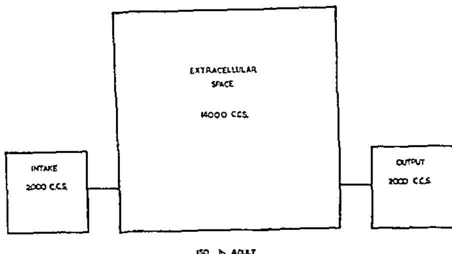


FIG. 31.—Diagrammatic illustration of the fact that the daily turnover of fluid in infants represents one-half of their extracellular fluid, as opposed to one-seventh in an adult (slightly modified from Gamble, 1950).

2. Water and electrolyte deficiency in seriously ill children who cannot take by mouth requires prompt, adequate and sustained replacement by parenteral routes; always remembering that early replacement of an observed loss is better than a heroic effort to correct an established deficit.

3. The recognition of water and salt retention in children is just as important as the recognition of water and salt deficit. Not only has the child's kidney a very limited capacity for excretion of excess water and salt but there is, as in the adult, a post-operative phase of impairment of the ability to excrete water and salt. Indiscriminate parenteral therapy in children can quickly give rise to œdema, with slow healing of both the intestinal anastomosis and the wound. In the case of a child the intravenous needle can be a very dangerous tool, on account of a temptation to give more than the calculated quantity and of the difficulty of giving parenteral fluid slowly enough.

Requirements. The basic daily requirements of fluid and salt vary with the age and size of the child. Tables VII and VIII give the main

FLUID BALANCE IN CHILDREN

by BRIAN W. WEBB, M.D., M.R.C.P., D.C.H.
Senior Registrar, Children's Dept., King's College Hospital

IN theory the management of fluid and electrolyte problems in infancy and childhood is no different from that in adults, but in practice their control is more difficult, as a child's fluid equilibrium is more delicately poised than that of an adult. In infancy and childhood the daily intake and output of water is relatively far greater than in an adult, and they have similarly smaller reserves on which to draw in an emergency, so that depletion develops more rapidly, and conversely over-loading is more easily produced. Rapid and dangerous changes in a child's state of hydration may be seen and they require the greatest care in their correction.

The relatively increased daily water output of infancy and childhood is due to alteration of both main channels of loss. In the first place the insensible loss is increased because of the more rapid respiratory rate and of the increased surface area in proportion to the body volume. In the second place the obligatory urine output is increased on account of the proportionately greater excretion of metabolic products and the smaller capacity of the child's kidney to concentrate urine.

This relative increase in daily output necessarily involves a similar increase in intake. Figure 31 illustrates the fact diagrammatically, and shows that the daily turnover of fluid in an infant constitutes one-half of his extracellular fluid as compared with only one-seventh in an adult. Bearing in mind that similar considerations apply, though to a lesser extent, in respect to salt, it is obvious that a significant disturbance in a child's fluid and electrolyte equilibrium can easily be caused even by a day without intake. If, in addition, there are gastro-intestinal losses or increased insensible loss from pyrexia, a situation of the utmost gravity can arise within a few hours. Without a doubt it is this extreme lability of balance which causes the main difficulty in maintenance and control of fluid disturbances in childhood and infancy. It will be helpful to consider the problem of fluid balance in childhood before proceeding to the very particular problems of the infant in the first weeks of life.

CHILDREN

Principles

1. Children undergoing simple surgical procedures do not need any supplementary water by parenteral routes.

Methods

1. *Intravenous fluid* is the most satisfactory way of maintaining parenteral fluid therapy. Its success in infants depends upon attention to detail. In larger children the usual sites, such as the forearm, may be chosen, but in smaller children it often proves necessary to "cut down" on a vein at the ankle or antecubital fossa, and insert a polythene catheter if parenteral therapy is required for several days. The ankle veins often prove unsatisfactory in practice on account of the fact that the patient is so small that the limb is hidden under the instrument trays and cannot be reached if adjustment of the catheter position is necessary during the operation. In small children and infants the scalp veins are easy to enter with a suitable needle,

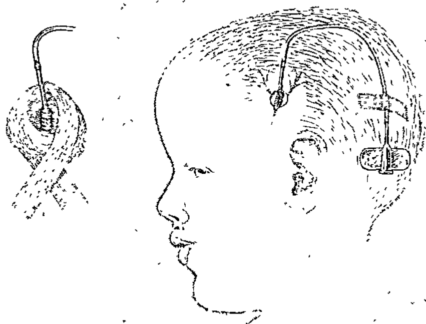


FIG. 32.—Diagram ill[illegible] bandage (4' × ½').

and if this is immobilised carefully, fluid can be given by this means for several days. Figure 32 shows how this may be achieved with a scalp vein needle attached to polythene tubing and immobilised by plaster of paris strips, or, alternatively, cotton wool soaked in collodion. Scalp vein transfusions are seldom used at the time of

TABLE VII

APPROXIMATE NORMAL NaCl MAINTENANCE ALLOWANCES PER DAY FOR RESTING UNSWEATING PERSONS

Size	*Sodium Chloride*	*Normal Saline*
Infants . .	1 gm.	125 c c.
Children .	3 gm.	350 c.c.
Adolescents and Adults . .	6 gm.	700 c c.

(From Butler and Talbot, 1944)

TABLE VIII

APPROXIMATE NORMAL WATER LOSSES AND ALLOWANCES* PER DAY FOR PERSONS OF VARYING SIZE NOT SUBJECT TO EXERCISE OR SWEATING

(1 kilogramme = 2 2 lb)

Size	*Water Loss*				*Usual Water Allowances*		
	Urine c c	*Stool* c c.	*Insensible* c c	*Total* c c	c c /person	c.c./Kg	oz./lb
Infant (2–10 Kg)	200–500	25–40	75–300†	300–840	330–1000	165–100	2 5–1 5
Child (10–40 Kg)	500–800	40–100	300–600	840–1500	1000–1800	100–45	1 5–0 7
Adolescent or Adult (60 Kg)	800–1000	100	600–1000‡	1500–2100	1800–2500	45–30	0 7–0 5

(From Butler and Talbot, 1944)

* Including water content and water of oxidation of food, which, under normal circumstances, except for infants, approximates the insensible water loss

† 1 3 c c per kilogramme per hour.

‡ 0 5 c c per kilogramme per hour

Note —Figures given for urine output are sufficient to excrete increased metabolites due to operation but do not provide for any decrease in renal concentrating power.

outlines of these needs compared with those of an adult. It should be noted that the figures shown for small infants refer to an infant fully established on a normal fluid intake and not to a newborn infant in the first week of life, which problem is considered separately at the end of this chapter. The calculated quantity may be increased to cover abnormal losses, such as excessive sweating, vomiting, intestinal suction, etc., and should be altered as necessary on the results of clinical observation at the bedside. The usual signs of over-hydration and under-hydration are easily recognised in children and can be confirmed by the results of daily weighing of the child. The importance of daily weighing as a reliable guide to alterations in the state of hydration of a child can hardly be over-emphasised.

time of operation and later, if the hæmoglobin level falls below normal, when the amount to be given may be calculated in the knowledge that an infant's blood volume is 100 ml/Kg. body weight. Plasma is often useful, especially if the plasma protein level falls below normal, and may be given to a total of 25 ml/Kg. body weight.

Congenital hypertrophic pyloric stenosis is an example of the problem of fluid balance in infants which is frequently encountered. The incidence of this condition has been estimated as 1 in 150 male infants and 1 in 775 female infants. It is, therefore, a fairly common illness and surgical treatment has become established as the treatment of choice. Its consistent success depends to a great extent upon the management of fluid balance; which involves the correction of pre-existing deficits and to a much smaller degree the maintenance of fluid balance for a brief period after the operation. In practice the problem presents in three different forms, each of which requires different management.

1. *Early Cases* where the child shows only a small deficit with a weight loss of less than 5 per cent.—and normal serum electrolyte levels. These children do well if they have one or two stomach washouts with plain water, or normal saline, until the washout is returned clean and they are then taken to the theatre for operation with an empty stomach.

2. *Moderately Severe Cases* where vomiting has been persistent for several days; these forming the great majority of cases. The weight loss may amount to 5–10 per cent. of body weight and the serum electrolyte levels show a hypochloræmic alkalosis with chloride levels of 90–95 mEq/l. and bicarbonate levels of 30–35 mEq/l. These children need parenteral fluids to correct their depletion. The fluid may be given subcutaneously or intravenously. If subcutaneous treatment is given it should be in the form of normal saline, or ½-normal saline in 2·5 per cent. glucose, 50–100 ml/Kg. every 12–24 hours. If intravenous treatment is given it should be in the form of ½-normal saline in 2·5 per cent. glucose at the rate of 150 ml./Kg./day. If it is necessary to continue for more than 24 hours the rate should be reduced to 100–150 ml./Kg/day and the strength of the saline reduced to 1/3 normal in 3·3 per cent. glucose for the subsequent period. When the weight loss has been restored and the chloride level raised above 98 mEq/l. the parenteral fluids may be withdrawn and the child taken to the theatre for operation a few hours later. Throughout this time oral feeding may be continued with expressed breast milk or half-strength, half-cream dried milk in small quantities (up to 3 ounces every 4 hours), but gastric washouts with plain water, or normal saline, should be carried out every 12 hours until the gastric residue is free from excess mucus. The stomach should always be washed out and left empty before the

the operation, as they may impede the freedom of the anæsthetist, but they are very useful if for some reason the "cut-down" transfusion has to be abandoned later. Once a scalp vein transfusion is set up and properly immobilised by the technique illustrated, the infant may be picked up and nursed with ease.

2. *Subcutaneous fluid* is valuable under certain circumstances, when the need for parenteral fluid therapy is limited in volume and in time. A good example is that of an infant a month old with pyloric stenosis and only mild depletion and hypochloræmia, who may need less than 100 ml./Kg. of ½-normal saline in 2·5 per cent. glucose to correct his fluid and electrolyte imbalance, and whose surgical treatment will rapidly remove any need for further parenteral therapy. The use of the technique is limited by the fact that the rate of uptake is slow and by the fluids that can be given, only normal saline or ½-normal saline in 2·5 per cent. glucose being suitable. Up to 100 ml/Kg. can be given into the thighs or axillæ over 4–8 hours and this can be achieved more easily and with even less discomfort to the baby if half an ampoule of hyaluronidase, equivalent to 500 International Units, is given into the injection sites (Gaisford and Evans, 1949). The child will usually sleep through this procedure.

3. *Rectal.* Small quantities of fluid may sometimes be given to children by continuous drip through a catheter inserted into the rectum. The method is not often used, as it proves unreliable and inaccurate.

Solutions. Complex solutions are widely used in pædiatrics, but it is fortunately true to say that it is seldom essential to use these solutions in surgical practice in a general hospital. 1/5 normal saline in 4 per cent. glucose, normal saline, and 5 per cent. glucose will cover most needs. The former solution should be used to cover basic requirements, and physiological saline should be used to replace abnormal intestinal losses. Five per cent. glucose may be used to correct pure water deficiency as indicated by clinical dehydration with normal or raised serum electrolyte levels.

In the rare instances where intravenous therapy has continued for several days, and intestinal losses have been heavy, hypokalæmia may retard recovery. In these cases, if there is a normal urinary output, KCl may be added to the solutions, but the concentration must never exceed 40 mEq/l. of fluid and the total given must not exceed 4 mEq/Kg. body weight in 24 hours. The addition of 1 gramme of KCl to a litre of fluid gives a solution containing approximately 13 mEq/l. of potassium. In the rare instances where severe acidosis fails to respond to therapy with glucose and saline, 1/6 Molar Sodium lactate (a disposable anion solution) may be given up to a total of 30 ml./Kg. body weight.

Blood can usefully be given both to replace estimated losses at the

time of operation and later if the hæmoglobin level falls below normal, when the amount to be given may be calculated in the knowledge that an infant's blood volume is 100 ml/Kg. body weight. Plasma is often useful, especially if the plasma protein level falls below normal, and may be given to a total of 25 ml/Kg. body weight.

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infant goes to the theatre. It is rarely necessary to submit an infant to surgical treatment until his depletion, or the greater part of it, has been corrected.

3. *Severe Cases* where vomiting has persisted for so long that the infant is already seriously ill with a severe state of depletion. The weight loss may be over 10 per cent. of body weight and the chloride deficit may be so severe that the serum chloride level has fallen below 90 mEq/l. In this case there is almost always a severe alkalosis and some depletion of both sodium and potassium. The same treatment should be given as for moderate depletion but intravenous treatment should be preferred to subcutaneous treatment. Once the urinary output has been restored small amounts of potassium should be added to the intravenous treatment for 12–24 hours. If potassium chloride is added at the dilution of 1 gramme per litre and solution is given slowly to a total of not more than 150 ml./Kg./day both the concentration and total dose will be within safe limits and usually effective in relieving potassium deficiency. If the alkalosis is so severe that tetany is threatened then more rapid correction of the alkalosis may be achieved by the addition of small quantities of a disposable cation solution (1/6 Molar Ammonium Chloride solution up to 30 ml./Kg. body weight).

If tetany actually occurs it can be relieved by the intravenous administration of 1 per cent. calcium chloride (5 ml./Kg. body weight given very slowly over 10–20 minutes). If the child has been desperately ill it may be wise to continue intravenous fluids during, and for a short time after, the operation, using 1/5 normal saline in 4 per cent. glucose for this maintenance therapy. In nearly every case, however, Ramstedt's operation relieves the obstruction of the pylorus at once and post-operative fluid balance can be maintained by oral feeding.

Post-operative treatment consists in oral feedings begun 4 hours after the operation, with 1 drachm of 5 per cent. glucose, the time between feeds, the volume of the feeds and the strength of milk in the feeds being steadily increased until the child is taking normal feeds of breast milk or half-cream milk every 4 hours 1 to 2 days after the operation. The total daily intake of milk 2 days after the operation should be at least 160 ml./Kg./day (2½ ounces/lb./day).

If vomiting occurs it will usually be controlled by a stomach washout and a reduction in the size and strength of the next feed.

The First Week of Life

Infants in the first few days of life not infrequently need urgent surgical treatment, usually for the correction of congenital abnormalities of the gastro-intestinal tract, such as œsophageal atresia,

imperforate anus, atresias of the small bowel and exomphalos. These newborn infants withstand surgical treatment very well, but a clear-cut schedule of fluid therapy must be established to tide them over until oral feeding can be established by the third to fifth day. It is important to remember that the newborn infant usually takes very little fluid until the breast milk begins to come in on the third day. It is not until the end of the first week that the infant is taking the full normal quantity of fluid, 160 ml./Kg./day (2½ ounces per pound per day). Because of this, great care must be taken to see that only small quantities of fluid are given over the first few days of life and the following schedule is suggested as one which has proved successful in a large number of cases. The need for surgical treatment in this group is usually rapidly apparent and as a result pre-existing losses are small and can generally be ignored.

An intravenous infusion is set up before the infant is taken to the theatre and for this purpose experience has shown that a "cut-down" with a polythene catheter inserted into a vein in the arm is most reliable.

For the first three days after operation fluid is given at the rate of 65–85 ml./Kg./day or 30–40 ml./lb./day. In the case of a 7 lb. infant this means giving about 250 ml. over the 24 hours, and requires most careful control by a nurse who must keep the infusion running at only 4–5 drops per minute. In practice it is found that movements of the babies can cause variations in the rate of flow. If an increase in the rate is allowed to occur it will almost inevitably result in excessive administration of fluid, for it is seldom possible to regulate the infusion later to a slower rate. For accurate control a graduated flask, showing 1 ml. divisions up to 100 ml., may be incorporated in the transfusion apparatus above the drip bulb.

For the first two days only 5 per cent. glucose is infused. On the third day 1/5 normal saline in 4 per cent. glucose is infused. If the infusion is still required on the fourth and fifth days it is likely that some complication has occurred and blood or plasma 25 ml./Kg. may be added to the 1/5 normal saline in 4 per cent. glucose, which is continued indefinitely if necessary. Every second or third day a further transfusion of plasma may be given with benefit, and serves the function of adding protein and potassium to the saline and sugar that the infant is already receiving. Biochemical control is desirable every second or third day, but it will be difficult to achieve unless the laboratory is equipped for micro-chemical methods of estimation of samples taken from the external jugular vein or from an ear prick. However, it is unlikely that any serious variations from the normal values will occur if the above schedule is carried out. If, however, there are severe intestinal losses these should be replaced by adding an equivalent volume of normal saline to the infusion, and if these

losses are continued for more than one day potassium should be added to the main solution as KCl 1 gramme/litre.

In the case of premature infants weighing 5½ lb. or less it is wise to refrain from using any saline at all, but rather to use 5 per cent. glucose in quantities not greater than 65 ml./Kg./day (30 ml./lb./day) for 4–5 days and to use plasma in small quantities to raise the serum protein which is usually low in premature infants. The plasma should be given as a half-strength solution in 2·5 per cent. glucose every second or third day to a total of 25 ml./Kg. (Gross and Ferguson, 1952).

In conclusion it must be clearly stated that the control of fluid balance in infancy and childhood involves a sustained effort on the part of the nursing and medical staffs. The nursing staff are called upon to maintain treatment by constant watchfulness and to keep clear and accurate records of the intake and, as far as it can be ascertained, the output of the children, as well as the daily weight of the infant. The medical staff are called upon to make regular assessments of the child's condition at the bedside and to issue clear and concise instructions for subsequent management in the light of the basic principles which we have tried to set out. Those who are willing to undertake this labour will be rewarded by seeing post-operative recoveries which would have seemed remarkable only a few years ago.

TECHNICAL CONSIDERATIONS

Most of the practical aspects of fluid and electrolyte problems lie within the realm of nursing care and are outside the scope of this monograph. But two particular points—namely fluid balance charts and intravenous "drips"—merit more detailed consideration, as the ease and efficiency with which difficult cases are handled can be greatly influenced by these two technical details. The rectal administration of fluids will not be discussed in any detail, not because it is thought an inefficient or unsuitable method, but because we have had little practical experience in its use. The rectal route is considered by many to be an uncertain and unreliable method of fluid administration, and it is certainly not, as it is often claimed to be, immune from the dangers of overloading. But in many units fluids are given routinely by rectal tube with certainty, and there is no doubt that with good technique this route of administration is quite satisfactory for the provision of the basic fluid intake. Similarly fluids can be given quite satisfactorily by the subcutaneous route, particularly if hyaluronidase is added to the solution, but this route is not satisfactory for the administration of large quantities of fluid. In our experience the greatest value of subcutaneous fluids is to supplement the oral intake of patients who for one reason or another cannot take enough by mouth. Thus, sometimes in the early days after the establishment of an ileostomy the patient may, on an oral intake only, fail to take enough water and salt by mouth and 0·5–1·0 l. of normal saline subcutaneously during sleep may benefit them greatly, and it is in circumstances such as this that subcutaneous fluids appear to be of greatest use.

Fluid Balance Charts

The lay-out of the charts for recording the data relevant to fluid balance is of great importance, as thereon largely depends the ease and rapidity with which the surgeon obtains the information vital to the making of many decisions. Undue stress can be laid upon the detailed arrangements of these charts, but in their design particular care should be taken to see that they provide the necessary data in a clear and simple form, and further that they can easily be filled in, even by the most junior nurse. Figure 33 shows charts designed with these objects in mind, and extensive experience has shown them to be both simple in use and helpful in the management of fluid problems of all types and complexity. Detailed observations regarding urine volume, amount of vomit or aspirate, etc., etc., are entered on

FLUID INTAKE & OUTPUT

Time	INTAKE			OUTPUT				
	By Mouth	Intra-venous	Other routes	Urine Volume	Chlorides or sp gr	Vomit or aspiration	Other routes	
8								
9								
10								
11								
12								
1								
2								
3								
4								
5								
6								
7								
Total								

B = Blood P = Plasma S = Normal saline G = Glucose,
1 pint = 560 ccs 1 oz = 28 ccs Insensible loss 500 ccs per 12 hrs

PLAN Balance

Date Name Ward

WEEKLY FLUID CHART

Name Age Ward

Date		INTAKE					OUTPUT			
		Oral	Intravenous Total	Intravenous Details	Other Routes	Total	Urine	Vomit or aspiration	Other Routes	Total
	Day									
	Night									
	Day									
	Night									
	Day									
	Night									
	Day									
	Night									
	Day									
	Night									
	Day									
	Night									
	Day									
	Night									

FIG. 33.—Fluid balance charts: that on the left is a 12-hourly chart for recording individual amounts of urine, etc., and that on the right a weekly chart for 12-hourly totals. Note the space on the 12-hourly chart for instructions. This chart is printed on both sides, so that it lasts 24 hours.

the 12-hourly charts, and at the end of each period the totals are entered on the weekly sheet. By this use of two charts, only one of which need be permanently retained, it is possible to obtain accurate information in respect of both the immediate situation and the overall trend of events, and this separation of 12-hourly totals from the hour-to-hour figures undoubtedly helps to avoid confusion. Of particular value is the provision on the 12-hourly sheet of a generous space for instructions; experience shows that many mistakes in handling arise from misunderstanding of the surgeon's instructions, and it is of great assistance to the nursing staff if these are clearly written on the sheet they are constantly using for recording urine measurements, etc. This form of charting does mean that the fluid requirements must be specified 12-hourly rather than 24-hourly, but in any complicated case this is essential, and all cases receiving intravenous fluids should be reviewed as often as this. Undue work is not caused by this system, for when patients are taking fluids normally by mouth, detailed records and instructions are not required. Ideally, all measurements should be recorded uniformly either in ounces or in millilitres, preferably the latter, as much confusion is caused when both units of measurement are used on the same chart. Unfortunately, the present education of nurses makes this aim almost unattainable; apparently unalterable custom decrees that oral fluids, vomit and urine output are measured in ounces, whereas intravenous fluids and aspirates are measured in cubic centimetres. A constant cause of error and exasperation would be removed if a single system of mensuration could be instituted in this branch of nursing.

Intravenous Transfusions

The comfort and confidence of a patient are greatly influenced by the skill and care with which intravenous drips are put up and maintained. The two essential requirements of a "drip" are that it should immobilise the patient as little as possible and that it should keep running for as long as possible. Nothing is more tiring for a patient than the constant changing of his drip, which is then maintained in position by cumbersome splinting.

The two common sites for insertion of an intravenous drip are the internal saphenous vein at the ankle, and the forearm veins. If the ankle is used, insertion of a cannula by the "cut-down" technique is almost inevitable, and in addition splinting of the ankle is required if the drip is to be kept going for any length of time. This latter method is particularly undesirable after operation, when active leg exercises are important in the prevention of decubitus thrombosis, and for both reasons this site should not be used for intravenous infusions unless no suitable arm veins are available. With respect to

the use of the forearm, the easiest veins for insertion of a needle usually lie across the wrist or elbow, but a needle cannot be secured in these situations without using splints, so that these sites should be used only when short transfusions of 0·5–1·0 litre are being given. In all cases where intravenous fluids are to be given for several hours or longer, the best place for insertion of the needle is the flat of the forearm (Fig. 34), and a suitable vein can nearly always be found in this position, particularly if venous congestion is produced by use of a light tourniquet and direct heat. In this position a "drip" can be securely inserted without immobilising the patient, and will run well for two or more days if properly cared for.

The actual type of needle or cannula used depends largely on the personal choice of the operator, but there appear to be few better instruments for this purpose than the West Middlesex needle (Fig. 35). When used for percutaneous insertion the trocar is withdrawn once the vein is entered, and the smooth cannula can then be slipped farther up the vein without risk of damaging the intima. In addition, if cutting down is necessary, the cannula is admirable for this purpose, so reducing to a minimum the equipment required. With regard to the size of needle or cannula used, the largest possible should always be inserted, as the rate at which a drip can flow is essentially governed by the bore of the needle, In a most interesting investigation into the hydrodynamics of transfusion, Melrose and Shackman (1951) showed that doubling the diameter of the cannula gave a 16-fold increase in rate of flow, whereas a 4-fold increase in pressure was required to double the rate. They also showed that tapering junctions within the system were desirable, otherwise the laminar flow is disturbed by turbulence, with marked slowing of the rate.

By far the commonest cause of a "drip" stopping is thrombosis in the vein around the needle. Neat, atraumatic insertion of the needle and occasional, rapid flushing of the tubing and cannula both help to avoid this occurrence, which is further prevented by the additional of heparin to the fluid used, as suggested by Martin (1944) The addition of 500 units of heparin to each 0·5 l. of fluid administered does not affect the recipient's clotting time, but materially delays the development of a clot in the immediate vicinity of the needle. For several years now we have added heparin in this amount to all intravenous fluids, and many hundreds of litres so treated have been given without complication.

Recently several workers have recommended the administration of intravenous fluids by means of a polythene catheter passed from a subcutaneous vein up into one of the major venous trunks, such as the superior vena cava, common iliac vein, etc. The advantage of this method lies in the fact that the infused fluid is rapidly diluted

in a large volume of blood, so that irritation of the vein wall is minimal and thrombosis is unlikely to occur. Erwin, Strickler and Rice (1953) report on 349 patients who received intravenous fluids by a polythene catheter passed from the basilic vein up into a large vein in the root of the neck; in the large majority of these cases the catheter was *in situ* for only 2 or 3 days, but in 1 patient it was retained for 46 days, and the authors state that in no case was there any serious or alarming local or general complication. We have reserved the use of this type of intravenous infusion to patients with anuria requiring hypertonic glucose solutions, and, more commonly, to patients who were going to require intravenous fluids for many days on end. We now have experience of the use of this technique in some 50 patients. From this experience we believe it is preferable to pass the catheter (polythene, size No. 2 or No. 3) via the basilic vein into the subclavian or innominate veins or the superior vena cava, rather than via the saphenous into the iliac veins or inferior vena cava. We have found this latter method liable to cause a femoral thrombosis, and whilst no permanent disabilities have resulted this occurrence is undoubtedly undesirable. Before inserting the catheter into the basilic vein its end should be carefully smoothed by friction, and the correct length approximately measured off against the patient to allow the tip of the catheter to be as near the auricle as possible. The actual insertion is simply done under local analgesia, and a variation of the rate of flow in time with respiration shows that the catheter is in one of the great veins. We have always added heparin (*vide supra*) to fluids given in this way. There is no doubt that using this technique a drip can be set up quite simply, which is very comfortable for the patient, which restricts his activities minimally, which will run for many days without trouble, and which can then be withdrawn easily. On many occasions we have kept such drips going for 8 or more days without trouble, but if there is any sign of irritation of the walls of the major veins it is wise to withdraw the catheter at once. This technique should not be used as a routine procedure, but should be reserved for selected cases requiring intravenous fluids for many days, and particularly those who require strong glucose solutions and other such fluids: in these circumstances the technique is a valuable assistance in treatment.

Many other technical problems arise in connection with the investigation and treatment of patients with fluid and electrolyte disturbances, and their solution depends largely on the personnel and equipment available. It is highly desirable that facilities should exist for the performance at all times of day of the more important and simple estimations required. Of particular value in this respect is a range of copper sulphate bottles (Phillips *et al.*, 1950) for the

rapid determination of the hæmoglobin and plasma protein concentrations by the method originally described by Van Slyke. An hygrometer for testing the urine specific gravity is, of course, part of the standard ward equipment, and an accurate method of determining the urine chloride concentrations should also be available. Serum electrolyte concentrations and other more complicated estimations require the facilities of a well-equipped laboratory.

A carefully considered routine and skilled technique are essential for ease in the handling of uncomplicated cases, and without their aid the diagnosis and management of patients with complex disturbances of fluid metabolism becomes hazardous and erratic. But, above all, the most outstanding requirement for success is unremitting concentration upon the part of the surgeon. Not only must all cases of difficulty be reviewed and assessed at frequent intervals, but care must also be taken to see that assistants and nurses realise their important responsibility in accurately measuring and recording all losses, and in carrying out scrupulously the instructions regarding intake. As with so many facets of surgical care, skilfully controlled teamwork is the key to success, and in its absence disaster can easily occur.

The majority of surgical patients present no problems in respect of the maintenance of water and electrolyte balance, but the minority develop serious disturbances of fluid metabolism either before or after operation. With our increasing knowledge of the physiology and pathology of the body fluids, most of these patients can be successfully treated if this knowledge is applied promptly and skilfully. "One may generalise by saying that if one will restore the patient to as near normal as possible before operation, maintain him thus during and after operation, not complicating his progress by needless treatment but unceasingly watching him for complications, there is an excellent chance that everyone needing operative treatment may be carried through to a successful convalescence" (Coller and De Weese, 1949).

Fig 34.—The best site for an intravenous drip is the flat of the forearm, as no splinting is required with a needle inserted here. The controlling tap should be close to the needle, to minimise the risk of air embolism.

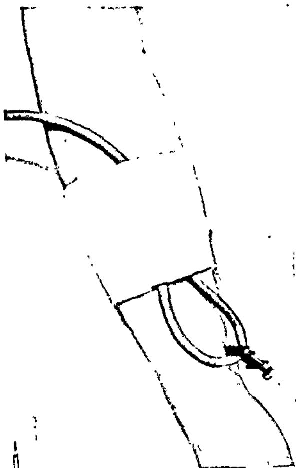








Fig 35.—A West Middlesex needle.

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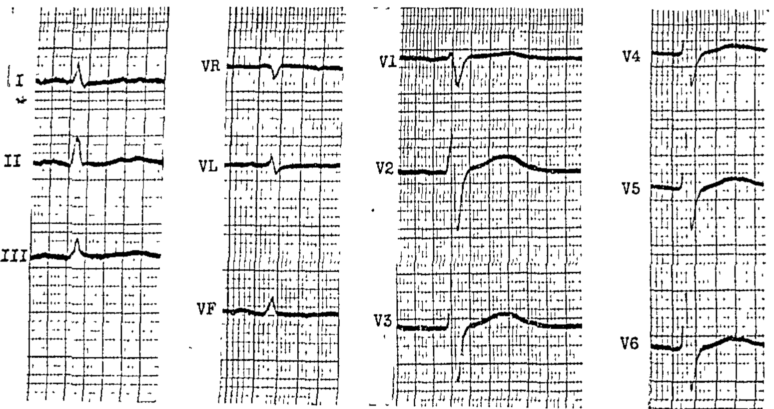


FIG. 37 —Case 4 23.ix. Serum K, 3·0 mEq/l The low, wide T waves fusing with U waves in the chest leads are characteristic of well-developed potassium deficiency.

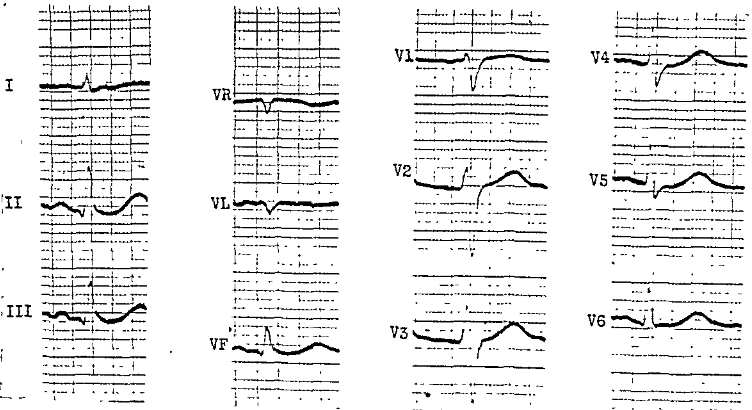


Fig. 36.—Case 4. 22 ix. Serum K, 3·3 mEq/l. The E.C.G. on admission shows a sagging depression of the ST segments in leads II and III, and widening of the T wave in the chest leads. These changes are consistent with *potassium deficiency*.

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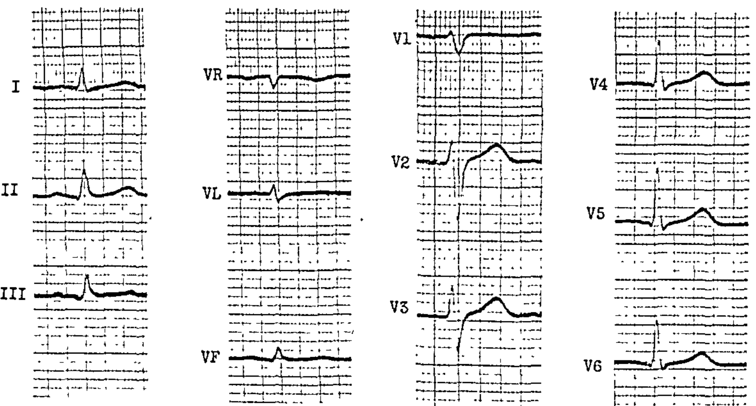


FIG. 38.—Case 4. 26 IX. *Serum K, 4·3 mEq/l.* The E.C.G. is now normal in all respects.

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APPENDIX A

TABLE SHOWING THE CHEMICAL AND IONIC STRUCTURE OF THE FLUIDS RECOMMENDED IN THE TEXT FOR INTRAVENOUS ADMINISTRATION

Solution	*Chemical Composition gm. per cent.*	*Ionic Composition mEq/l.*
Normal saline . .	0·9 per cent. NaCl	160
Saline-potassium . Mixture . . .	0·225 per cent. NaCl 0·3 per cent. KCl	Na 40 K 40 Cl 80
Potassium Chloride .	0·3 per cent.	40
M/6 Na Lactate . .	2·2 per cent.	165
M/6 Ammon. Chlor. .	0·85 per cent.	165

These solutions are *commonly made up in distilled water. It is preferable to* make up the saline-potassium mixture in 2·5 per cent. dextrose, thus making the solution isotonic.

APPENDIX B

CASE HISTORIES

THE following case histories are included to illustrate various points in the practical management of fluid and electrolyte disturbances, and for this purpose only the essential clinical details are included. Reference has been made to these reports in the text but, in addition, comments are attached to each case report to stress the points of importance and significance. The histories are deliberately reported in note form, in the interests of brevity.

Case 1. Mr. C. O'B., *æt.* 68 years.

29.ii.52 Admitted with chronic retention of urine with overflow, due to benign prostatic enlargement. Bl. urea, 120 mgm. per cent. Suprapubic catheterisation (Riches' Instrument) performed; technically, recovery uneventful.

6.iii.52 Bl. urea, 100 mgm. per cent.

12.iii.52 Bl. urea, 82 mgm. per cent.

27.iii.52 Bl. urea, 62 mgm. per cent.

	U. Vol.		*U. Vol*		*U. Vol.*		*U. Vol.*
29.ii	40	7 iii	95	14.iii	104	21.iii	101
1.iii	114	8.iii	71	15.iii	114	22.iii	87
2.iii	85	9 iii	67	16.iii	98	23.iii	93
3 iii	109	10 iii	71	17.iii	102	24.iii	111
4 iii	146	11.iii	70	18 iii	122	25.iii	97
5.iii	78	12.iii	103	19 iii	141	26 iii	82
6 iii	93	13.iii	66	20.iii	105	27.iii	91

Urine volume, per 24 hours. Volumes measured in ounces. The figure for 29.ii represents 12-hour output only.

Urea Clearance (Van Slyke)

12 iii.52 Urea clearance, 53 per cent. normal.
Urine urea, 0·79 gm. per cent.

Water Concentration and Dilution Test

After 12 hours with no food or fluid intake, the patient passes urine, then again 60 min. and 120 min. later. 1500 ml. of water are then drunk in 20 min., and all the urine passed in the next three

hours is collected. In a patient with normal kidneys, the specific gravity of at least one of the first three specimens exceeds 1025, and during the last 3 hours the urine volume will exceed 1000 ml.

11.iii.52 Sp. gr. of first three specimens 1004, 1009, 1014 respectively.
Total urine volume in last 3 hours, 350 ml.

12.iii.52 Sp. gr. of first three specimens 1010, 1007, 1006 respectively.
Total urine volume in last 3 hours, 300 ml.

Comment

A case illustrating the effects of severe renal damage.

Note:

(1) The marked inability of the kidneys to secrete a concentrated urine, thus making the minimum daily obligatory volume high (see Fig. 4).

(2) The consequent very slow fall in blood urea, despite a persistently high urine output. Compare the rapid fall in bl. urea in Case 3, who, being able to pass a urine of specific gravity 1027, cleared 32·5 gm. urea from the body in 24 hours, whereas this man only cleared *10·0 gm. in the period 29.ii to 6.iii. (No allowance made for* intake of N.)

(3) The value of the urine concentration and dilution test in emphasising the degree of renal failure. Imagine that the information re-bl. urea, urine output, etc., prior to 11.iii was not available, and the value of this test becomes more obvious.

(4) Clinically this man showed no signs of derangement of water balance, yet any major operation would have been fraught with danger.

(5) The importance of maintaining a high urine output in this man, to prevent increasing nitrogenous retention.

Case 2. Mrs. McK., *æt.* 41 years.

28.vi. 2 a.m. Admitted with a strangulated umbilical hernia of 30 hours' duration: showed moderate but definite signs of extracellular dehydration, though vomiting had not been marked; Hb, 111 per cent.; Ht, 47 per cent.; Pl.Pr. 7·5 gm. per cent. Pl Na 149 mEq/l.; Pl.Cl 100 mEq/l. Urine sp. gr. 1035. Urine Cl 34 mEq/l. I.V. drip set up and operation performed (gut resection not required). Up to 11 a.m. 1·5 l. of normal saline and 1·5 l. of 5 per cent. glucose were given intravenously.

28.vi. 11 a.m. Clinically satisfactory; skin elasticity normal now. Hb, 84 per cent. Pl.Pr. 5·75 gm. per cent.; Ht, 39 per cent.; Pl.Na 147 mEq/l.; Pl.Cl 106 mEq/l. Urine volume, 180 ml. of sp gr. 1023, and Cl concentration 17 mEq/l.

29.vi. 10 a.m. Steady improvement clinically: no clinical signs of dehydration. Since 11 a.m. 28.vi has received 3·0 l. normal saline and 2·0 l. 5 per cent. glucose. Blood figures essentially unaltered; urine volume, 2320 ml. of sp. gr. 1010–1001; urine chloride concentration, 5 mEq/l.

Comment

A case illustrating the difficulties of replacing, during the post-operative period, a pre-existing water and salt deficit. At the time this patient was treated our understanding of the normal post-operative changes was minimal, and the intake higher than we would now advise.

Note:

(1) The marked fall in hæmoglobin, hæmatocrit and plasma protein figures between 2 a.m. and 11 a.m. (28.vi), due largely to the correction of the extracellular deficit, and possibly also to some water retention as a result of the metabolic response to surgery.

(2) After the administration of 3·0 l. of fluid intravenously in 9 hours the urine volume was only 180 ml., with a specific gravity of 1023. Owing to the performance of an operation during this period, it is impossible to say to what extent this oliguria with a high gravity is due to inadequate replacement of the deficit or is a manifestation of the post-operative impairment of water excretion: probably it is almost entirely due to the latter.

(3) Similarly the polyuria between 11 a.m. 28.vi and 10 a.m. 29.vi may have been due either to excessive replenishment of the deficit, or to the excretion of excess water retained as the result of the temporary impairment of water excretion after operation.

(4) Despite the administration of large quantities of saline intravenously the urine chloride concentration steadily diminished, though there were no abnormal losses; this is not indicative of increasing salt deficiency, but is a manifestation of the post-operative inability to excrete salt.

(5) During the period recorded, both the urine specific gravity and chloride concentration figures, on their face value, gave highly misleading indications as to the treatment required, as they were influenced largely by the characteristic post-operative changes, and not by the progress of replenishment. Replenishment during the post-operative period must be controlled essentially by clinical findings.

Case 3. A. T., *æt.* 31 years.

Transverse colostomy performed on 31.x.51 for tuberculous colitis. During 10.xi and 11.xi 51 started to vomit, and during 12.xi.51 his

hours is collected. In a patient with normal kidneys, the specific gravity of at least one of the first three specimens exceeds 1025, and during the last 3 hours the urine volume will exceed 1000 ml.

11.iii.52 Sp. gr. of first three specimens 1004, 1009, 1014 respectively.
Total urine volume in last 3 hours, 350 ml.

12.iii.52 Sp. gr. of first three specimens 1010, 1007, 1006 respectively.
Total urine volume in last 3 hours, 300 ml.

Comment

A case illustrating the effects of severe renal damage.

Notes

(1) The marked inability of the kidneys to secrete a concentrated urine, thus making the minimum daily obligatory volume high (see Fig. 4).

(2) The consequent very slow fall in blood urea, despite a persistently high urine output. Compare the rapid fall in bl. urea in Case 3, who, being able to pass a urine of specific gravity 1027, cleared 32·5 gm. urea from the body in 24 hours, whereas this man only cleared 10·0 gm. in the period 29.ii to 6.iii. (No allowance made for intake of N.)

(3) The value of the urine concentration and dilution test in emphasising the degree of renal failure. Imagine that the information re-bl. urea, urine output, etc., prior to 11.iii was not available, and the value of this test becomes more obvious.

(4) Clinically this man showed no signs of derangement of water balance, yet any major operation would have been fraught with danger.

(5) The importance of maintaining a high urine output in this man, to prevent increasing nitrogenous retention.

Case 2. Mrs. McK., *æt.* 41 years.

21.vi. 2 a.m. Admitted with a strangulated umbilical hernia of 30 hours' duration; showed moderate but definite signs of extracellular dehydration, though vomiting had not been marked; Hb. 111 per cent.; Ht. 47 per cent.; Pl.Pr. 7·5 gm. per cent. Pl.Na 149 mEq/l; Pl.Cl 100 mEq/l. Urine sp. gr. 1035. Urine Cl 34 mEq/l. I.V. drip set up and operation performed (gut resection not required). Up to 11 a.m. 1·5 l. of normal saline and 1·5 l. of 5 per cent. glucose were given intravenously.

22.vi. 11 a.m. Clinically satisfactory; skin elasticity normal now. Hb. 84 per cent. Pl.Pr. 5·75 gm. per cent.; Ht. 39 per cent.; Pl.Na 147 mEq/l; Pl.Cl 106 mEq/l. Urine volume, 180 ml. of sp. gr. 1023, and Cl concentration 17 mEq/l.

29.vi. 10 a.m. Steady improvement clinically: no clinical signs of dehydration. Since 11 a.m. 28.vi has received 3·0 l. normal saline and 2·0 l. 5 per cent. glucose. Blood figures essentially unaltered; urine volume, 2320 ml. of sp. gr. 1010–1001; urine chloride concentration, 5 mEq/l.

Comment

A case illustrating the difficulties of replacing, during the post-operative period, a pre-existing water and salt deficit. At the time this patient was treated our understanding of the normal post-operative changes was minimal, and the intake higher than we would now advise.

Note:

(1) The marked fall in hæmoglobin, hæmatocrit and plasma protein figures between 2 a.m. and 11 a.m. (28.vi), due largely to the correction of the extracellular deficit, and possibly also to some water retention as a result of the metabolic response to surgery.

(2) After the administration of 3·0 l. of fluid intravenously in 9 hours the urine volume was only 180 ml., with a specific gravity of 1023. Owing to the performance of an operation during this period, it is impossible to say to what extent this oliguria with a high gravity is due to inadequate replacement of the deficit or is a manifestation of the post-operative impairment of water excretion: probably it is almost entirely due to the latter.

(3) Similarly the polyuria between 11 a.m. 28.vi and 10 a.m. 29.vi may have been due either to excessive replenishment of the deficit, or to the excretion of excess water retained as the result of the temporary impairment of water excretion after operation

(4) Despite the administration of large quantities of saline intravenously the urine chloride concentration steadily diminished, though there were no abnormal losses; this is not indicative of increasing salt deficiency, but is a manifestation of the post-operative inability to excrete salt.

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Case 3. A. T., *æt.* 31 years

Transverse colostomy performed on 31 x.51 for tuberculous colitis. During 10.xi and 11.xi.51 started to vomit, and during 12.xi.51 his

condition deteriorated rapidly. X-rays showed dilated small gut, ? due to obstruction, ? to ileus. No evidence of strangulation.

12.xi.51 6 p.m. Pale, sweating, lethargic. Retching and vomiting, ? due to obstruction, ? to salt deficiency. Pulse poor in volume, 110/min. B. P. 80/50 (variable). Extremities clammy; skin elasticity markedly diminished; tongue moist; eyeballs soft. Complaining of thirst, and limbs hypotonic. Urine output, 4 oz. in last 10 hours; sp. gr., 1027; chlorides, absent.

Hb, 110 per cent.; Bl. urea, 114 mgm. per cent.

Pl.Na 128 mEq/l.*; Pl.Cl 78 mEq/l.*; Pl.K specimen hæmolysed.

Comment.—Severe extracellular salt water deficiency, probably of about 4·0–6·0 l. Main loss probably "concealed" (i.e. into gut), as vomiting has not exceeded 50 oz. in last 24 hours. Potassium deficiency also present, but correction not advisable until extracellular deficiency largely replaced and urine output restored.

To receive by 8 a.m. tomorrow 3·0 l. of normal saline and 0·5 l. 5 per cent. dextrose.

13.xi.51 10 a.m. Much improved. Not so lethargic; extremities, warm and dry; pulse, 100/min.; B. P., 120/55. Skin elasticity only slightly diminished.

Urine output, 540 ml. plus (incontinent once); sp. gr., falling to 1010; chlorides, absent.

Hb, 87 per cent.; Bl. urea, 105 mgm. per cent.

Pl.Na 126 mEq/l.*; Pl.Cl 85 mEq/l.*; Pl.K 3 mEq/l.*

During night 1 litre aspirated via Ryle's tube.

Comment.—During next 12 hours to receive (*a*) basic intake 1·0 l. 5 per cent. dextrose, 0·5 l. N. saline; (*b*) vol.-for-vol. replacement 1·0 l. N saline; (*c*) for pre-existing deficiency, further 0·5 l. of NaCl and KCl mixture, 40 mEq.K. Total 3·0 l.

8 p.m. During 12 hours fluids given as ordered; urine output, 700 ml., but chlorides still absent: 300 ml. aspirated via tube.

Comment.—Deficit still not replaced: to receive in next 12 hours (*a*) basic intake 1·5 l. 5 per cent. dextrose; (*b*) vol.-for-vol. replacement 0·5 l. N saline; (*c*) for pre-existing loss, 0·5 l. N saline. Total 2·5 l.

14.xi.51 8 a.m. Clinically improved; skin elasticity normal; B. P., 120/55. Still complaining of some thirst.

Urine output 1700 ml. in last 24 hours, but sp. gr. has risen to 1015 and during night intake short by 0·5 l. of dextrose ordered. Urine Cl 7 mEq/l.

Hb, 87 per cent.; Bl. urea, 40 mgm. per cent.
Pl.Na 134 mEq/l.*; Pl.Cl 87 mEq/l.*; Pl.K 3·4 mEq/l.*

Comment.—Deficit largely replaced, but not enough water for excretory purposes, as shown by rising urine sp. gr. Is now ready for potassium administration in larger quantities, and as abdominal condition improving this to be given by mouth, but also continue intravenous fluids.

15.xi.51 During last 24 hours has received 2·0 l. of saline and 3·0 l. 5 per cent. glucose intravenously, together with some water and pot. citrate by mouth. No clinical signs of depletion. Urine output, 2020 ml. with sp. gr. 1003, and chloride concentration, 53 mEq/l.
Hb, 78 per cent.; Bl. urea, 25 mgm. per cent.
Replacement of deficit completed: further progress of case was uneventful.

Comment

A severe case of extracellular deficiency, with peripheral circulatory failure and azotæmia; also some potassium deficiency, but this not very marked. The estimations marked with an asterisk were not available until 24 hours after the blood specimen was taken, and were not used in controlling treatment.

Note:

(1) Replacement was controlled on the basis of clinical observations, hæmoglobin estimations and the urine figures. Note the method of calculating requirements each 12 hours.

(2) The urine chloride concentration was one of the last features to return to normal, showing its value as a "fine adjustment" in controlling extracellular replacement.

(3) The raised bl. urea resulting from dehydration, and its steady fall as the urine output was restored.

Case 4. F. S., *æt.* 54 years.

A man with complete pyloric obstruction due to carcinoma of the stomach. Admitted with marked dehydration, with alkalosis. For 9 days before operation the patient was treated with intravenous fluids and gastric suction; the replacement of the water and sodium chloride deficits presented no unusual problems. He also had potassium deficiency, and the balance studies and E.C.G. tracings are shown to illustrate the following points:

(1) Potassium deficiency was diagnosed on admission (see E.C.G., Fig. 36), but no potassium could be given until the urine output was restored, during which time he lost a further 45 mEq. K.

(2) The E.C.G. on 23.ix shows the potassium deficiency to be more marked, and the changes seen are characteristic (Fig. 37). The V leads clearly show depression and prolongation of the ST complex, with the appearance of U waves. Following administration of K, the E.C.G. rapidly returns to normal (Fig. 38).

(3) The potassium balance shows a marked retention when this ion is given. Note the characteristic fall in urine K excretion for the first few days after its administration (Fig. 39).

(4) For 9 days this man received intravenous fluids, and the cumulative negative nitrogen balance shows the size of the deficit developed and illustrates the magnitude of the nutritional problem in such cases. The N output on 22.ix is broken down into its two components, and throughout the ratio between gastric and urine loss was similar (Fig. 40).

Case 5. C. W., *æt.* 63 years.

29.iii.51 *Gut resection for strangulated femoral hernia.*

30.iii.51 General condition good: towards evening gastric aspirate began to increase.

31.iii.51 Developed severe ileus, with aspirate from nasogastric tube exceeding 5·0 l. in 24 hours. Treated by (1) Provision of basic daily intake of 1·0 l. N saline and 2·0 l. 5 per cent. glucose; (2) Volume-for-volume replacement with N saline of observed losses; (3) KCl solution given also.

Ileus, with aspirate in excess of 4·0 l. per 24 hours, continued for four days, and then relented. Oral feeding re-established on sixth day. Throughout this period treatment given as outlined above

Comment

The sodium and chloride balances are illustrated to emphasise the following points (see Figs. 41 and 42):

(1) Note the size of the daily loss—over 500 mEq of Na and Cl daily during the three days of worst ileus. Failure to replace a loss of this size would soon cause death.

(2) Losses of this magnitude emphasise the necessity for measuring abnormal losses, otherwise replacement becomes completely haphazard.

(3) Note the retention of Na and Cl both greatly in excess of the deficit on 30 iii. This retention, largely due to the post-operative impairment of salt excretion, was accompanied by very low urine sodium and chloride concentrations, which are *not* indicative, under these circumstances, of a salt deficit. This case demonstrates clearly that during the post-operative period the urine chloride concentration is not an accurate guide to salt intake.

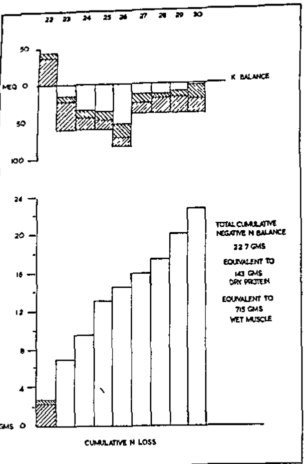


Fig. 39.—Potassium balance from Case 4. Note the negative balance during the first day of treatment, followed by a marked retention when potassium is given. Note also the fall in urinary excretion of potassium when the ion is given. The demonstration of retention of potassium following its administration is the only absolute proof of the existence of a potassium deficiency.

Fig. 40.—Data from Case 4, showing the cumulative negative nitrogen balance. Throughout the nine days of observation this man was on intravenous fluids and received no protein.

Note the large negative balance accumulating day by day. The nitrogen loss on the first day is broken down into its gastric and urinary components, and throughout the ratio between these two was similar.

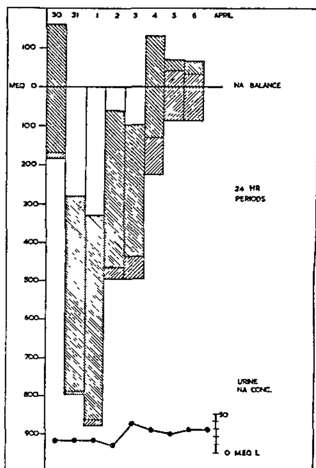


FIG 41.—Sodium balance from Case 5—see p. 132.

(4) In this case the salt intake was excessive, owing to (*a*) too big a basic intake; (*b*) the fact that the fluid lost was hypotonic—av. Cl concentration 105 mEq/l., as opposed to 160 mEq/l. in saline, the replacement fluid. But this patient made an uneventful recovery, illustrating the practical efficacy of the simple volume-for-volume method of replacement of observed losses. On the fourth and fifth days this man developed slight sacral and ankle œdema, which would probably have been avoided had the basic intake of salt not been excessive.

Case 6. Mrs. M. A. O., *æt.* 71 years.

A case of recurrent carcinoma of the cervix, with vesicovaginal fistula and bilateral hydroureter and hydronephrosis. Bl. urea, 27 mgm. per cent. Pl.Na 138 mEq./l.; Pl.Cl 102 mEq/l.; Pl.K 4.8 mEq/l.

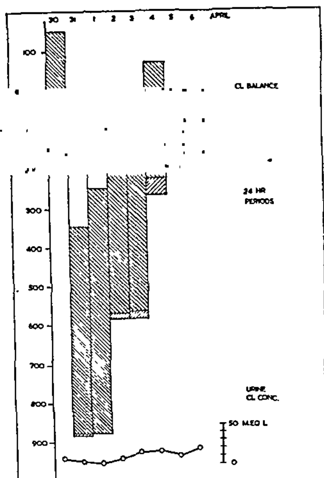


FIG. 42.—Chloride balance from Case 5—see p. 132.

19.x.52 Bilateral transplantation of ureters into bowel.

By 22.ix patient had a distended abdomen and was vomiting, and this continued for 4 days. Throughout the period 19.ix–26.ix the patient received no K intravenously, and little beyond water by mouth. Vomiting never appears to have exceeded 500 ml. in 24 hours.

26.ix.52 Laparotomy—no mechanical obstruction found.

27.ix.52 No change—Miller-Abbott tube passed.

28.ix.52 No change—distended, silent abdomen: Bl. urea, 56 mgm. per cent.

1.x.52 Some deterioration: now apathetic, and lethargic with muscular hypotonicity. Pl.Cl 87 mEq/l.; Pl.Na 136 mEq/l.; Pl.K 3·5 mEq/l.

2.x.52 Very apathetic and sleepy; muscular hypotonicity marked; abdomen distended and silent. No peripheral œdema. Pl.K 2·7 mEq/l.

Between 8 a.m. (2.x) and 8 a.m. (3.x) given a litre of KCl (80 mEq) intravenously, apart from glucose, etc.
During that time loss only 11 mEq, showing a retention of 69 mEq K.

3.x.52 Slightly more alert and stronger, but belly still distended and silent. Bl. urea, 31 mgm. per cent. Pl.Na 140 mEq./l.; Pl.Cl 100 mEq./l.; Pl.K 3·2 mEq/l.
Given a further 80 mEq K intravenously during next 24 hours.

4.x.52 Much improved: more alert and muscle tone improving. Bowel sounds present. Pl.K 4·1 mEq/l.
Given a further 40 mEq K intravenously and started on fluids by mouth, with pot. citrate.

5 x.52 Further marked improvement; full oral feeding re-established.

Comment

A very characteristic case of potassium deficiency. For 13 days the potassium intake was zero or negligible, and during this time there were (*a*) increased urinary losses, due to two operations, (*b*) abnormal losses from the alimentary tract. The clinical picture was highly characteristic, with marked apathy, muscular hypotonicity and chronic ileus. E C.G. tracings, which are not reproduced, and the plasma K concentration confirmed the diagnosis. Note the rapid and dramatic response to the administration of 80 mEq.K per 24 hours, and note that the condition could have been prevented by the addition of K to the basic intake. Note also the mild degree of alkalosis accompanying the hypokalæmia.

Case 7. Mrs. E. L., *æt.* 69 years.

20.x.52 Admitted with perforated gastric ulcer of 10 hours' duration.
General condition very poor—B. P. 100/70.
Given 1·0 l. of plasma intravenously before operation.

21.x 52 B. P., 90/60.
Intravenous intake, 2·0 l. 5 per cent. glucose and 0·5 l. N saline.
Urine output, 300 ml

22.x.52 B P., 100/70
Fluid intake, 2·5 l. 5 per cent. dextrose, 0·5 l. N saline.
Urine output, 340 ml.

23.x.52 B. P., 160/100; Bl. urea, 125 mgm. per cent.; Pl.K 5·2 mEq/l.
During the previous 48 hours there had been no appreciable abnormal losses, and calculations showed the water intake

to exceed the output by at least 3·0 l. Clinically the patient was fully hydrated, with slight sacral œdema. Taking into account the prolonged fall in B. P., the oliguria, rising bl. urea and high plasma K, a diagnosis was made of acute tubular necrosis, probably not severe. Administration of the correct oral mixture was impossible, so the only treatment given was to restrict fluids to 1 litre of 5 per cent. dextrose intravenously. During the course of the day a spontaneous diuresis developed, the patient passing 1460 ml. of urine with a sp. gr. *1010–1012*.

24.x.52 Fluids again restricted to 1 litre of 5 per cent. dextrose, and diuresis continued; 1650 ml. of urine passed, with sp. gr. of later specimens falling to 1002. During the ensuing 3 days the patient was given 3·0 l. of water and 4·5 gm. of salt daily, and during this time the urine output was within normal limits and the bl. urea fell to 31 mgm. per cent. Thereafter normal oral feeding was reverted to.

Comment

A mild but typical case of acute tubular necrosis, due to a prolonged fall in blood pressure. This case clearly illustrates the importance of early diagnosis; after 48 hours' oliguria with a normal fluid intake, this patient had retained 3 l. of fluid, and both the bl. urea and plasma potassium showed significant rises. Fortunately a spontaneous diuresis developed quickly, so that this delay was not serious, and the retained water, potassium and urea were quickly excreted. When anuria is complete the diagnosis is obvious, but it is equally important to institute early treatment in cases such as this.

Case 8. Mrs. G. V., *æt.* 45 years.

26.iii.52 L. inguinal colostomy for rectovaginal fistula, due to carcinoma of cervix.

27.iii.52 When asked to see this patient 24 hours after operation, a presumptive diagnosis of acute tubular necrosis had been made, as no urine had been passed since operation. But during the previous 24 hours the intake had been only 1 litre, whilst apart from insensible loss the patient had vomited 700 ml. In addition, clinical examination showed slight signs of dehydration. It was decided that the anuria was due to inadequate intake; 2·0 l. of 5 per cent. dextrose and 0·5 l. N saline intravenously in 12 hours rapidly restored the urine output.

Comment

A simple case of anuria due to inadequate intake, but illustrating clearly the necessity for diagnosing accurately the cause of anuria or oliguria during the post-operative period. Compare with Case 7, in which there was marked oliguria with a normal intake. This case also emphasises the value and importance of simple observations and deductions before embarking on complex biochemical estimations.

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